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A CONTRIBUTION TO THE COMPARATIVE STUDY OF CONVULSIONS.

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1. *Introductory.*

WHEN I first began the investigation of nervous diseases I supposed, as most other physicians then did, and as perhaps most still do, the seat of epilepsy to be the medulla oblongata. But I soon came to the conclusion, that the cerebrum is the seat of epileptiform seizures (convulsion beginning unilaterally). I supposed the particular parts diseased to lie within the region of the middle cerebral artery; that rough localisation was before I had the advantage of the experimental researches of Hitzig and Ferrier. Later on, I supposed that, as I crudely put it, epilepsy (the epilepsy proper of authorities) depended on discharges of convolutions "at a greater distance from the motor tract" than epileptiform seizures did.[1] I believed then, that no variety of convulsion in man arose from any sort of change below the cerebrum proper. But very lately I have come back to the belief, that *some* convulsions (I do not call them epileptic) in children (and, I suspect some in adults which are *called* epileptic)

[1] Study of Convulsions, 'St. And. Med. Graduates' Trans.,' vol. iii. 1870.

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depend on lesions of the pons or medulla oblongata. The fits in children to which I especially allude are "inward fits," otherwise called laryngismus stridulus; they are "respiratory convulsions."

Of course I knew long ago that Brown-Séquard had demonstrated that in guinea-pigs a *liability to* convulsions could be experimentally produced (in ways everybody knows), and that the fits could be artificially brought on when the brain proper had been taken away. And I knew that Kussmaul and Tenner had produced convulsion by rapidly bleeding rabbits when the brain proper had been removed. It is plain that the convulsions in these animals do not depend on the cerebrum. Moreover there are the most valuable experiments of Westphal, another way of producing a liability to convulsions in guinea-pigs. But I supposed, erroneously I now think, that no sort of convulsion occurred *in man* from any kind of disease lower than the cerebrum proper. I thought the reason why convulsion occurred in certain lower animals deprived of the cerebrum, and not in man from disease of parts lower than the cerebrum, was explainable by the manifest great differences in the degree of evolution of the nervous systems of those animals from that of the nervous system of man. Seeing that the convulsions in the animals just mentioned were demonstrably owing to changes in the medulla oblongata, or pons, or both, I tried to show why such convulsions occurred in these lower animals and not in man.[1] Having now modified my belief on this matter, I come to attach even a greater value than I once did to the researches of Marshall Hall, Brown-Séquard, Kussmaul and Tenner, and Westphal. This, by way of mere preliminary. I now think, as above stated, that there are three classes of fits in man. I go on to speak more particularly of the three.

2. *The Scale of Fits.*

I believe (1) epileptic fits (epilepsy proper) to depend on "discharging lesions" of parts of the highest level of evolution, and (2) epileptiform seizures to depend on "discharging

[1] 'West Riding Asylum Reports,' vol. iii. p. 342. "On Evolution of Nervous Centres."

lesions" of parts of the middle level of evolution. Both (1 and 2) are cerebral convulsions. Both, to my thinking, are "cortical." (3) Inward fits (respiratory convulsions) and some other fits depend, I submit, on discharges beginning in parts of the lowest level of evolution.

3. *The Three Levels of Evolution of the Central Nervous System.*

I now give a brief outline of the hierarchy of the nervous centres. It is admittedly imperfect; it is incomplete, too, by omission of the sympathetic chain, peripheral ganglia, and ganglia of the posterior roots of the spinal nerves. (Strictly, we ought to speak of four levels, the periphery being the real lowest.) It will be seen that the scheme is not after the morphological divisions, spinal cord, pons Varolii, &c., but is, especially, according to degree of indirectness with which centres represent parts of the body, and is thus an anatomico-physiological scheme. The motor nuclei of the pons Varolii and medulla oblongata are some lowest motor centres as much as the anterior spinal horns are some others.

(1) The lowest level of evolution, or series of lowest centres, is pretty much what Marshall Hall called "the True Spinal System," which, according to him, especially consisted of spinal cord, medulla oblongata, and pons Varolii. The nuclei for ocular muscles in the floor of the aqueduct of Sylvius are, no doubt, the topographically highest known lowest motor centres.[1]

The lowest sensori-motor centres represent all parts of the body, animal and organic, "from nose to feet," in simplest, &c., combinations.

Besides the centres we have spoken of, anterior and posterior spinal horns and their homologues higher up, and besides Clarke's visceral column and its lower and higher representatives, there are what are called "regulating centres," —micturition centre, vaso-motor centre, respiratory centre, &c. It may be that these are simply re-combinations of elements of the nuclei mentioned. I think that these centres are products of internal evolution.

[1] There is no impropriety in this expression, nor in such a one as "two lowest centres," since "lowest centre" is a proper name.

(2.) The middle level of evolution consists of Hitzig and Ferrier's motor centres, and of Schäfer and Victor Horsley's trunk centres, and also of Ferrier's sensory region.[1]

The middle sensori-motor centres re-represent all parts of the body, organic and animal, from nose to feet, in more complex, &c., combinations.

(3.) The highest level of evolution consists of parts of the brain in front of the middle motor centres (frontal or pre-frontal, lobes), highest motor centres, and of parts behind the middle sensory centres (occipital lobes), highest sensory centres.

The highest centres re-re-represent all parts of the body, organic, and animal, "from nose to feet," in most complex, &c., combinations.

The lowest level of central evolution being evolved out of the periphery (epi- and ento-) represents all parts indirectly, but yet most nearly directly of all the levels; the middle, evolved out of the lowest, represents (re-represents) all parts doubly indirectly; the highest evolved out of the middle, represent (re-re-represent) all parts triply indirectly.

The highest centres, the climax of the evolution, are in popular language the "organ of the mind." I again insist, as I have done for many years, that this "organ" is as certainly of sensori-motor constitution as is the lumbar enlargement (some lowest centres). But the constitution of the highest centres differs vastly in degree from that of the lowest centres. They (*a*) represent most indirectly (re-re-represent) (*b*) literally all parts of the body having nerves (as the lower levels do), but they (*c*) represent all parts in most numerous and most complex, &c., combinations. Just as any state of consciousness is a state of a whole person psychical, so the correlative activity is of nervous arrangements representing a whole person physical (the whole organism). When a person is having a particular mental state, there are occurring physically, of course, activities of his highest centres. But

[1] Since Hitzig and Ferrier are not agreed on details of motor representation by the cortex, it will be seen that by using the expression "Hitzig and Ferrier's motor centres," I do not pretend to demarcate exactly the middle and highest motor-centres.

what I urge is that the nervous arrangements then engaged represent his body, and some parts of it most especially.

I believe that the cerebellum also represents all parts of the body, but in ways different from the cerebrum. It will be seen that the lowest level of evolution is at once lowest cerebral and lowest cerebellar.

4. *The Comparative Study of Convulsions; Varieties of Lowest Level Fits.*

One object is to make a comparative study of the three classes of convulsions or fits depending on discharges on the three different levels of evolution,—lowest, middle and highest, levels of evolution. Speaking roughly, there are supposed to be lowest, middle and highest "level fits."[1] Several things qualifying a direct comparison and contrast of the three classes of fits will be spoken of later.

An "inward fit," or respiratory convulsion, is but one variety of fits on the lowest level, just as convulsion beginning in one hand is but one variety of epileptiform seizures (middle level fits), and just as convulsion ushered in by an "epigastric sensation" is but one variety of epilepsy proper (highest level fits). Moreover what we call respiratory convulsion should in many cases, since the convulsion becomes universal, be called respiratorily beginning convulsion. In each class of fits the convulsion is universal if the discharge be severe enough. Ordinary spasmodic asthma in the child or adult is a respiratory convulsion, and is also a lowest level fit. Both it and inward fits are, I submit, owing to sudden, &c., discharges beginning in some part of the respiratory centres. Rigor is another lowest level fit, dependent on discharge beginning in some part of the vasomotor centre. I suppose, as implied in some foregoing remarks, that the fits induced by Brown-Séquard and Westphal in guinea-pigs, and by Kussmaul and Tenner in rabbits, are lowest level fits in those animals. I shall consider later whether, as I have said I suspect, some fits

[1] In still other words, which must not be taken literally, there are "least evolved," "higher evolved," and "most evolved" fits. Thus a severe epileptic seizure is a "more evolved fit" than a severe epileptiform seizure is, even when in each the convulsion is universal. I shall no longer use such expressions, for strictly the three classes of fits are three different dissolutions *being effected*.

in the adult called epileptic are really epileptic (epilepsy proper), or are lowest level fits—fits analogous to those occurring in Brown-Séquard's guinea-pigs. But I ought to say now, that I fear I shall not be able to make out anything like a demonstration on this matter. In any case we should have to consider the vast differences in the degree of evolution of the nervous centres in man and in the guinea-pig.[1]

It may be well to say very explicitly, what is, however, repeating part of the foregoing, that I do not believe epilepsy, the epilepsy proper of nosologists, and epileptiform seizures to be owing to any sort of lesion on the lowest level of evolution, although of course the motor centres on this lowest level are secondarily engaged in epileptiform seizures, and tertiarily in epileptic seizures.

"Inward fits," or respiratory convulsions, some lowest level fits, are the main subject of this paper; epileptic seizures, highest level fits, have been spoken of at the outset for the sake of order, and will be considered again in comparison and contrast with "inward fits," and other lowest level fits.

5. *Inward Fits.*

"Inward fits," or laryngismus stridulus (respiratory convulsions), occur mostly in children under one year of age, not often after two. Laryngismus stridulus occurs especially, Niemeyer says, during the first year of life; Charles West found that out of thirty-seven cases, thirty-one occurred between the age of 6 months and 2 years. Meigs and Pepper write:[2] "Of thirty cases selected indifferently from our practice, and from authors in which the age is given, thirteen were 6 months or less of age, eleven between 6 months

[1] Since writing this sentence, I have seen fits in a boy whose case is, I think, very like the cases of Brown-Séquard's guinea-pigs. A boy of seven, falls suddenly to the ground when his head or face, either side, is touched; his face flushes, his eyes turn up and to the right, his respiration stops, and there is a sudden jerk of his limbs. Since his fits began a few months before the age of two and a half years (when he had "an ordinary epileptic fit"), as he has imperfect left hemiplegia, and for other reasons to be given when the case is reported, the supposition of pretence is about as reasonable as that Brown-Séquard's guinea-pigs should pretend to have fits when the epileptogenous zone is touched.

[2] 'Diseases of Children,' 7th edition.

and 1 year, four between 1 and 2 years of age, one of 2 and one of 4 years of age; so that of the thirty, twenty-four were under 1 year." "Laryngismus seldom attacks children more than a year old."[1] Henoch writes that it is "confined almost exclusively to the period between the 6th and 24th months. I have hardly ever observed it at a later age, but have seen cases as early as the fifth or sixth week."[2] What is the significance of this limitation by age?

6. *The Infant's Nervous System.*

The lowest level of evolution is probably nearly the whole of the new-born infant's developed nervous system; the higher levels, in-so-far as they are not yet organised, are masses of "nervous stuff" and not centres proper; they are almost a fœtal part of the new-born infant. No doubt development goes on with increased activity in the higher two levels after birth; the nervous masses, as we called them, will be rapidly becoming middle and highest centres. But their development will be far behind that of the lowest ("earliest") level. "Inward fits" are rare under six months of age, when the lowest level itself will be very imperfectly developed; but probably the main reason why these fits do not often occur until the age of six months is that rickets is rare before that age.

According to Soltmann, the convolutions in the "motor region" (I call this region the middle motor centres) in new-born dogs, cats and rabbits is unexcitable at birth, and epileptiform seizures cannot be provoked in them by excitation of that region. Albertoni has confirmed these observations. Franck and Pitres write of Albertoni's researches: "Il résulte de cette série d'expériences que l'épilepsie par la faradisation de l'écorce du cerveau n'a pu être provoqué chez un chien de treize jours, tandis qu'elle s'est produite chez les animaux âgés de dix-neuf jours et plus." Soltmann's conclusions have, however, been recently disputed by Dr. Joseph Pareth, who says the dog's cortex *is* excitable in the first days after birth. Still we cannot but suppose that the higher

[1] Ringer, 'Therapeutics,' 10th edition, p. 67

[2] 'Lectures on Diseases of Children,' p. 70.

centres must, in new-born animals, be a long way behind the lowest in their degree of development.

The infancy of a dog in comparison with that of man is of slight duration. The higher the animal, the longer its infancy. Man's infancy is the longest of all. The following quotation from Fiske is of profound importance to the medical evolutionist in many ways.

"The gulf by which the lowest known man is separated from the highest known ape consists in the great increase of his cerebral surface, with the accompanying intelligence and in the very long duration of his infancy. These two things have gone hand-in-hand. The increase of cerebral surface due to the working of natural selection in that direction alone has entailed a vast increase in the amount of cerebral organization that must be left to be completed after birth, and thus has prolonged the period of infancy."[1]

Much is said of the "great excitability" of the nervous system of infants. I should not speak so generally, but would say that the lowest level of evolution is in the new-born infant almost the whole of its then developed nervous system, and thus almost the sole part there is to be excitable. Putting this otherwise, the lowest level is, the younger the infant, the more nearly *its* highest level; it is indeed more nearly its highest and lowest at once, although, later in life, on full development of its middle and highest nervous masses into centres proper, that level will be the real lowest. It itself in the infant will be at once imperfectly developed and actively developing, and thus *naturally very excitable.* The parts most actively developing on the infant's lowest, his then nearly highest, level will be the centres for organic parts; the centres for animal parts will be far behind them in degree of development. No doubt the order of degree of development of the centres for the three great organic functions, although nearly contemporaneous, will be, both before and after birth, digestive, circulatory, and respiratory; the respiratory will be, of the three, the most actively developing in infancy. The lowest centres for the three systems are seated in that part of

[1] 'Destiny of Man,' p. 54. For a full account of Fiske's opinions on the importance of prolongation of infancy, see his 'Cosmic Philosophy,' vol. ii.

the lowest level which is, morphologically, the medulla-oblongata, and as Lauder Brunton, in some very important remarks, says: "the medulla oblongata in the vertebrata may be looked on as a lower and more fundamental centre than the brain or spinal cord."[1]

Another side of some of the foregoing statements is that the higher levels being in the infant little organised (being there morphologically, but not being there functionally to any great extent) the lowest level will be less "controlled" or less "kept down" than in older persons. *Pari passu* with the later development, or evolution, of the higher levels, the lowest will be more and more "kept down." Putting this otherwise; the process of evolution is not only an "adding on," but is thereby, at the same time, a "keeping down;" the newly added (properly the higher and newer *evolved out of* the lower and older) controls its lower. Using popular language, we gradually get above our mechanical selves as evolution of our middle and highest centres (and especially their internal evolution) proceeds.

Here I refer to Soltmann's important researches on imperfect development of inhibitory arrangements in very young dogs, cerebral, he thinks, as well as spinal. If Soltmann's observations are correct, they are a very important contribution to the doctrine of evolution of the nervous system. The younger the infant, the more of a mere ordinary reflex mechanism will be what there then is of its nervous system; the less check there will be of one part by another. The accelerating cardiac centres will develop ahead of the cardio-inhibitory centres; the respiratory ahead of its checking nervous arrangements, the vaso-motor ahead of the vaso-dilator nervous arrangements.

7. *Recapitulation.*

So now we find that "Inward fits" occur in the very young, in those persons that is, whose lowest level of evolution is the most developed and yet most actively developing level, and which, from the comparatively little development of higher centres, is little controlled. This is saying that what we call the infant's lowest centres are its *then* nearly

[1] 'Pharmacology,' &c., p. 197.

highest centres or highest and lowest at once. "Inward fits" occur in those persons whose *then* nearly highest centres are *naturally very excitable.* Bearing in mind Soltmann's observations, we add that these nearly highest centres (which later in life will be the lowest centres) are more nearly ordinary reflex centres, are less checked in their actions and interactions by inhibitory nervous arrangements. But why have some and not other infants "inward fits"? In other words, what is the determining cause of such discharges of the infant's "naturally very excitable" respiratory centres as produce, not respiratory movements, but respiratory convulsions? That inward fits nearly always occur in the rickety is now (Elsässer, Jenner, Gee) accepted doctrine. But what is the relation? Henoch writes: "All attempts fail to explain the connection between rachitis and spasm of the glottis." It is then somewhat rash in me to submit an explanation. The question is, "By what steps does rachitis bring about sudden and excessive discharges of the respiratory centres in infants? What is the intermediary between rickets and Inward fits?" As a preliminary, some general remarks on nervous discharges must be made.

8. *Digression of Nervous Discharge.*

All movements are the results of nervous discharges (or liberations of energy by nerve-cells), but in the case of convulsion there are sudden excessive and rapid discharges; *such* discharges of any motor centre do not produce movements proper.[1] Besides developing movements strongly, they develop many together—produce that contention of many movements which we call convulsion. Any motor centre suddenly, excessively and rapidly discharging is, for the time being, a "convulsive centre." What I call "discharging lesions" in epileptic and epileptiform seizures are small persistent "con-

[1] Many years ago Moxon remarked, "muscle is only in degree less nervous than nerve." There is, in severe epileptic fits, a sequence of discharges of four levels of motor evolution—highest, middle, and lowest nervous centres and of "muscle centres"—muscles being in one aspect, in accord with Moxon's remark, nervous centres; in another aspect they are glands. I continue, however, to speak of three levels of evolution.

vulsive centres." A "discharging lesion" (or part of a motor centre become a "convulsive centre") is a local persisting hyperphysiological state of nerve-cells induced by some pathological changes, which changes lead to increased, but yet, I think, inferior nutrition.[1]

It is to be insisted on that convulsion, in any case, is nothing whatever else than a sudden excessive and rapid development of the *normal* movements which the centre, suddenly and excessively discharging, represents. The great thing to bear in mind is, that such discharges lead not only to excessive, but also to contemporaneous development of many movements, so that the name "movements" is no longer properly applicable to the motor affection produced, and we call it convulsion. Let any motor centre be discharged strongly enough, and rapidly enough, and we have convulsion of the parts the centre represents.

There is, of course, something more. Since a "discharging lesion" is a physiological fulminate (normal cells of a centre having become a quasi-parasitical hyper-functioning part of it), the sudden excessive and rapid discharge of its *highly* unstable elements upsets the equilibrium of (discharges) anatomically connected nervous arrangements of normal instability; normal elements which, in the case of a "destructive lesion," serve towards compensating a loss, are in the case of "a discharging lesion" compelled to co-operate in an excess (co-operation in excess is compensation inverted). So it is always to be understood, when not stated, that not only are the movements of the parts most especially represented by cells constituting a "discharging lesion" developed, but that the movements of other parts also, represented by normal cells which are (by collateral and downward currents) compelled to discharge, are developed. (The discharges in both cases being sudden, &c., what we just called the development of movements, is really the "running them up" into convulsion.) Hence from sudden and rapid discharges of a small part of the highest centres (in epilepsy proper) we may have

[1] I have been said to have put forward the "theory of discharges" as the *pathology* of epilepsies. I have really tried hard to show that certain pathological processes only *induce* "discharging lesions" which are *hyper-physiological* states.

from wide collateral and downward discharges universal severe convulsion (co-operation in excess).

Convulsion, although very different from a series of normal movements, is not something altogether *sui generis*. I do not now believe, as I once said, that all metaphysic has died out of the expression "attacked by convulsion;" it is not always thoroughly realised that a convulsion is simply a brutish development of many of the patient's ordinary movements. A severe epileptic convulsion, to take that case for illustration, is nothing whatever else than a sudden, excessive, rapid, and consequently a nearly contemporaneous, development of many of the patient's common-place daily movements—of movements of the eyes in looking; of the face in its various expressions; of articulatory movements; of manipulatory and of other movements of the arms; of movements of the trunk and legs in walking; of digestive, respiratory and circulatory movements, &c. But from such developments there is no looking, smiling, talking, &c., because all the movements which are developed, being nearly contemporaneously developed, are "run up" into a muscular fight. Speaking figuratively, we may say that the epileptic discharge is trying to develop all the functions of the body excessively, and all at once; a severe fit is a fairly successful attempt. Let me give a very simple illustration. If there be a centre for locomotion, then during slight sequent discharges of its elements in health, there is walking or running; but if very many of those elements were to discharge suddenly, rapidly and excessively, the man walking or running would not go faster; on the contrary, he would be stopped, would be stiffened up into a tetanus-like attitude by the *contemporaneous* development of many locomotor movements.[1]

We must not speak of "disorderly discharges," but simply of degrees of discharges above the normal—of degrees of suddenness, excessiveness, and of rapidity of discharges. Coming close to our particular topic, we say that whilst normal

[1] It will be seen that I do not believe that an attack of mania ever replaces an epileptic convulsion; if cells of the nervous arrangements discharging so as to produce movements in mania were to begin to discharge so suddenly, &c., as nerve-cells do in an epileptic fit, the maniacal movements would be "run up" into convulsion.

discharges of the respiratory centres produce a sequence of respiratory movements, sudden excessive and rapid discharges of those centres produce a respiratory convulsion, which is a sudden excessive and contemporaneous development of, and therefore a strong contention of, normal respiratory movements.

9. *The Determining cause of sudden, &c., Discharges of Respiratory Centres in Inward Fits.*

It is agreed upon that the normal stimulus to the respiratory centres is venous blood. It has long been recognised that rapidly induced asphyxia in animals produces a certain kind of convulsion, a respiratory one. I believe the determining cause of the sudden excessive and rapid (convulsion-producing) discharge of the respiratory centres in cases of "inward fits" to be an excess of their "natural stimulant," venosity of blood. There is no persistent "discharging lesion," as I believe there to be in epileptic and epileptiform seizures. The normally "very excitable" respiratory centres are occasionally goaded into excessive activity. It is not necessary for me to deny, and I do not deny, that besides being naturally very excitable, the centres developed in the infant, and especially those most actively developing, are *morbidly* over-excitable in rickets and in some other diseases. But I make no use of that hypothesis. But how comes about this excess of the "natural stimulant"—excess of venosity in the rickets?

Mentioning the circular shape of the young child's chest (its respiration being chiefly diaphragmatic), I remark next that "inward fits" occur, not only in persons with such an imperfectly developed respiratory apparatus, but, as is well known, most often in those of them who, from rickets, have abnormally soft ribs. The ribs being abnormally soft in the rickety, and thus not "holding out" during the descent of the diaphragm, the efforts of that muscle are more or less neutralised. Thus there is in young rickety children a condition for venosity (which may in part account for the great sweating in these subjects). Further, the attacks of laryngismus occur chiefly at night when, from sleep, there is still less perfect respiratory

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action and thus a condition for still greater venosity. A crying fit may bring on a paroxysm (as, anticipating, laughing may bring on an attack of spasmodic asthma in an adult). Henoch[1] writes in his articles "Spasm of the Glottis," "In fact, many characteristics of the affection can be seen in a *healthy* child who, in the midst of a severe crying spell, suddenly 'holds the breath'; *i.e.* lies with the head thrown back, face dark red, somewhat cyanotic, respiration interrupted, and rigid limbs. The excessive crying and the excitement appear to cause spasm of certain respiratory muscles, which disappears, as a rule, after a few seconds." I submit that the thing first caused by the crying in such cases is super-venosity, and that this, over-stimulating the respiratory centres, causes respiratory spasm. Henoch then goes on to speak of *abnormal* conditions: "attacks [of laryngismus] may occur during complete quiet, and *even on awakening from sleep;* but even then it is favoured *by every respiratory exertion*, viz. crying, drinking, eating as well as by psychical influences, anger and fright." The parts I have italicised in the second half of the quotation seem to me in favour of my hypothesis.

Great venosity of the blood will much over-stimulate the "naturally very excitable" respiratory centres in infants, and thus produce respiratory convulsion.[2]

No doubt the respiratory centres are also acted on indirectly through the vagi, the venous blood stimulating the endings of these nerves in the lungs. For when the vagi are cut across, respiration is slower, but yet deeper. Stimulating the central end of one of the vagi, quickens respiratory movements; indeed great stimulation so accelerates the rhythm, that it ceases to be rhythm, ordinarily so-called; there is tetanus of the diaphragm, and standstill of respiration in an extreme inspiratory phase. Here we see that, increasing the rapidity of the discharges of the respiratory centres fuses the respiratory movements into convulsion.

[1] Op. cit., p. 70.

[2] Possibly, in so far as the higher levels are developed in infants, sleep, which in all persons is, among other things, the going out of function of the highest nervous arrangements (a normal dissolution), leaves the respiratory and other lowest level centres less controlled, and thus more excitable than during waking.

Of course no one can ignore that there is very great venosity of blood in the paroxysm of laryngismus, but my contention is that too great venosity is the first thing, that it precedes any part of the paroxysm. Whilst healthy breathing depends on slight venosity—the respiratory centres acting in healthy states from some slight deficiency of oxygen, or possibly, as some think, from stimulation by carbonic acid,[1] too great venosity will stimulate the respiratory centres so overmuch as to produce, not a sequence of normal respiratory movements, but that excessive development and contention of them which is convulsion—will produce arrest of respiration by fixation of the respiratory apparatus. In healthy respiration the inspiratory part is chief; expiration being to a great extent recoil from inspiration. But even if we could conceive that the inspiratory movements were alone suddenly, &c., developed, the chest would be more or less fixed, fixed in a high inspiratory attitude. In dyspnœa, the expiratory movements come into greater prominence.

When there has been first set up any degree of spasm of any part of the respiratory apparatus, the venosity will become greater still, and thus there will be a rapid multiplication of effects. There are degrees of "inward fits" from that local convulsion signified by "crowing inspiration" (perhaps after temporary complete closure of the glottis), to universal convulsion probably consequent on prolonged closure of the glottis, or on partial closure of it with also fixation of other parts of the respiratory apparatus. The supposition is, that the more rapid the initial discharges (that is, the more elements of the respiratory centres suddenly discharging at once, and thus the greater the rate of transfer of energy), the more is it likely for the convulsion to become universal.

That the convulsion, even when universal, is not owing to discharge of higher levels (not of the so-called motor region,

[1] Brunton writes (Pharmacology, &c., Ed. i. p. 202): "It is uncertain whether the stimulation which the venosity of the blood produces is due chiefly to the absence of oxygen or to the presence of carbonic acid. Possibly also it may be due to the products of imperfect combustion in the venous blood. Or all these three causes may share in the stimulation, though to what extent each does so is not known. According to Bernstein, want of oxygen appears to stimulate the inspiratory, and the presence of carbonic acid to stimulate the expiratory centre."

middle level, at least) is proved by the fact, that asphyxia renders the "motor area" of the cortex inexcitable.[1]

10. *Universalisation of Convulsion beginning Respiratorily.*

Supposing, then, the first sudden, &c., discharge in Inward fits to be of the respiratory centres in seizures becoming universal, the universalisation of the convulsion may be owing to secondarily induced discharge of other centres on the lowest level. Nearly all the muscles of the body must on demand, so to speak, serve in respiration. This implies that the respiratory centres, whilst representing some movements of the nose, larynx and chest nearly directly, represent also movements of nearly all other parts of the body with various degrees of greater indirectness; means that they can "call on" other motor centres to act with them in a certain order; means that their discharges, when strong enough, "find their own" in other centres—flow in lines which are of least resistance *to them*. It is not then necessary to suppose that there are big permanent respiratory centres for all parts of the body, but to hold that there are centres representing most especially the respiratory apparatus, from great over-action of which centres other centres, not ordinarily respiratory, can be called out as reserves, called upon to suspend their own individual duties to serve respiratorily, to join in the fight for breath. The term "centre," in any case, is an arbitrary expression.[2]

[1] It may be said that some clinical facts show that great venosity of the blood, in adults, "excites the brain." In some cases of emphysema with bronchitis, the patient may be delirious at night. To take a stronger case. A man was recently admitted into the London Hospital under Mr. McCarthy's care, for injury of the spine, which reduced the patient to diaphragmatic respiration. He became delirious, when he was evidently undergoing asphyxia. For my part I cannot see how these facts show that venosity *excites* the brain. I should say that the venosity accounted for the negative half only of the patient's condition when delirious—for the physical condition answering to his defect of consciousness—that it put out of use some of the highest nervous arrangements of the highest centres. I submit that the positive part of the condition, the delirium proper, was the outcome of raised activity of lower nervous arrangements of his highest centres, consequent on loss of control. We remarked before that Evolution is not only an "adding on," but a "keeping down" also; so in the case of Dissolution from venosity just mentioned, the "taking off" of the highest nervous arrangement is a "letting go" of the lower.

[2] For admirable remarks on the term "Centre," see Lauder Brunton, 'Pharmacology,' &c., 1st ed., p. 199.

I refer to remarks made in the section *Digression on Nervous Discharge* on secondary discharges of normal elements consequent on a primary sudden, &c., discharge of highly unstable elements.

It may perhaps be that the same cause, super-venosity, acts directly on other lowest centres, discharging them after discharging more or less of the respiratory centres. Indeed there certainly is action on some other lowest centres: for in artificially produced asphyxia there are discharges of the vaso-motor and sweat-centres, as well as of the respiratory centres.

The presumption is that there are the two effects; that universalisation of the convulsion depends partly on discharges spreading from the primarily discharging respiratory centres to other centres on the lowest level, and partly on elements of these other centres being also discharged by the direct action of over-venous blood upon them.[1]

In the foregoing is a detailed acknowledgment of what was stated generally, in saying that the convulsions in the respiratory fits of children sometimes become universal. There are effects produced by asphyxia in many centres, if not in centres "all along the line" of the lowest level. In children we see turning up of the eyes; asphyxia produces sweating, raised arterial tension; there are carpo-pedal contractions as well as the severer universal convulsion. Hence severe respiratory seizures, there being universal effects, are more thoroughly comparable and contrastable with severe epileptiform and epileptic seizures, in each of which also there are universal effects, than the too limited name "respiratory" I give to them implies.

If venosity in the rickety causes the respiratory fits we call laryngismus in infants, other causes of venosity in very young children should do the same.

[1] What we have so far called *the* respiratory centre is only the dominating respiratory centre. If the spinal cord be divided just below the medulla oblongata, respiration usually ceases entirely. But if strychnia be injected into the blood before the division, respiratory movements continue to some extent, and begin again if strychnia be injected after the division.—Lauder Brunton, op. cit. p. 201.

11. *Respiratory Convulsions in Laryngitis of Young Children.*

I refer to convulsions in young children, subjects of laryngitis ("spasmodic laryngitis"); they are respiratory convulsions, but may become universal. I suggest that the age of the patients, the fact that there is a manifest impediment to respiration at the gateway of the respiratory apparatus, and the occurrence of the attacks mostly at night (in sleep), point to an over-stimulation of the respiratory centres by too great venosity. If so, the hypothesis that attacks of laryngismus are similarly caused is favoured. "Spasmodic" laryngitis usually occurs in children under two or three years of age; the attacks usually occur suddenly during sleep, and next day the child may be seemingly well except for a little hoarseness and cough; the attacks are sometimes mistaken for laryngismus.

12. *Treatment of Inward Fits.*

Let us now see if the results of treatment favour the hypothesis put forward. It is good practice not to let the child who is the subject of laryngitis sleep too long at a time —not to let it remain too long in a condition favouring venosity; I suppose the same applies to laryngismus stridulus, to which morbid affection I confine further remarks on treatment.

It is understood, of course, that attention is given to diet, digestion and state of the bowels. No one fails to treat the rickets, the most approved medicine being cod-liver oil. The good effect of the highly valued remedy, cold sponging, favours my hypothesis; manifestly it will stimulate the respiratory centres. So do the good results of carrying the child out even in cold weather, and change from town to country air. The several remedies will tend to reduce venosity. Now for drugs, more especially in the treatment of what we may call laryngismus itself. The best remedies should, on the hypothesis, be those which stimulate the respiratory centres. Eustace Smith writes:[1] "Of special drugs, musk and belladonna are most useful [in laryngismus stridulus]. The former can be given to a child of twelve months old in doses of one-third of a

[1] 'Diseases of Children,' p. 273.

grain every six hours, and will be found to have a powerful influence in checking the tendency to spasm. Belladonna, to be of service, must be given in sufficient doses. A baby twelve months old, will take well fifteen drops [of the tincture] three times in the day." Musk[1] is a stimulant to the respiratory centre, as is also belladonna. It is well-known that children tolerate large doses of belladonna; bearing in mind Soltmann's researches, this may be because in them it acts more as a stimulant, or rather that the parts it can render negative (inhibitory and secretory nervous arrangements) are not largely present in the infant to be paralysed.

What is said of the beneficial effect of the stimulating action of belladonna on the respiratory centres is not inconsistent with the hypothesis, that great excess of the "natural stimulus" (venosity) produces the fits; for to keep respiration going on actively will prevent great venosity. But anything which makes the respiratory centres act excessively, causes so rapid a succession of impulses from them to respiratory muscles, as to fix the chest in one persistent inspiratory condition, and brings about next similar impulses to expiratory muscles. Thus there results a fight in which the chest is still in infinite effort, as two well-matched wrestlers are at a certain stage of their contest, the full power of each being developed.

Chloral is a remedy for fits of laryngismus lauded by high authorities. It is the best remedy for a rapid succession of fits of any kind—for putting an end to the seizures, that is. It is not, I presume, actually curative of the morbid condition inducing them. It may be given when fits of laryngismus are frequent.

Now for treatment after the paroxysm. When the fit is over, the child usually comes round spontaneously. But supposing that after a severe attack respiration no longer goes on. The respiratory centres are exhausted, or what remains of them unexhausted is not enough to produce respiratory movements; the child is seemingly dead. There is paralysis of the respiratory apparatus after respiratory convulsions, just as there may be of a limb after an epilepti-

[1] Brunton's 'Pharmacology,' &c., p. 988.

form convulsion which had affected that limb first and most.[1] Here we must bear in mind that, after asphyxia fits produced in lower animals, the heart, as a rule, continues to beat for a short time after respiration has stopped. Hence if luckily present when a child is seemingly dead after a fit, we should give him a chance of recovery by artificial respiration. We should not waste time to see whether the heart has or has not stopped. The following quotation[2] justifies the above remark, and is of value in several other ways. Brunton is speaking of asphyxia fits artificially produced in lower animals. The first paragraph in the quotation refers to the paroxysm, the second to the after-paralytic stage, the third is directly relevant to what was just said on attempts to restore infants apparently dead after their respiratory convulsion.

"As the blood becomes venous, the activity of the respiratory centre increases, the respirations becoming quicker and deeper, and the accessory respiratory muscles are thrown into action. This condition is called *dyspnœa*. Finally, the excitement extends to all the muscles of the body, and we get general *convulsions*, which have generally an opisthotonic character. The eyeballs very often protrude during these convulsions, and the blood-pressure rises greatly from stimulation of sympathetic and vaso-motor centres in the medulla.

"After the convulsions cease, the animal usually lies motionless, and the heart, as a rule, continues to beat for a short time after the respirations have ceased.

"The excessive venosity of the blood in this condition has paralysed the nerve centres; but if artificial respiration be now commenced, and the blood becomes gradually aërated, the conditions just described are again passed through in the reverse order; convulsions first reappearing, then dyspnœa, next normal breathing, and if the respiration be pushed far enough, apnœa."

I say nothing further here of carpo-pedal contractions which so often complicate laryngismus; their interpretation is difficult. If we take a mere general view of them, as "spasms,"

[1] I believe there to be paralysis after epileptic fits also; but, so far as I know, no one agrees with me in this supposition.

[2] Brunton, op. cit. p. 202.

there is no difficulty; but the question is, "Why are the most terminal parts of the limbs so specially subject to spasm in this disease?"

13. *On Convulsions in some other Morbid Affections of Young Children.*

Besides convulsion in rickety infants, and in those infants whose blood-aëration is more acutely in arrears from catarrhal laryngitis, I suppose that in some other diseases in which there is great venosity in very young children there are respiratory convulsions. Probably convulsions in young children who have congenital heart-disease are of that nature.

Eustace Smith writes,[1] dealing with congenital heart disease; "Convulsions are very common, especially in infants; and startings and twitchings during sleep are seldom absent, whatever be the age of the patients.

"Nearly one-half of the cases die before they have completed the first year, and two-thirds before they are two years old. Death often occurs in a convulsive fit; and infants usually die in or directly after such a seizure."

We have convulsions in some cases of whooping-cough, if indeed the ordinary attacks are not allied to convulsion. Judging from what Henoch says,[2] I should say that some convulsions in patients who have pertussis are very closely like the severe convulsions of laryngismus. The parts I have italicised in the following quotation are particularly interesting in this regard—the first as to age; the second as to contractions, resembling, if not identical with, carpo-pedal and other contractions. Henoch calls attention to "the unfavourable significance of those paroxysms [of whooping-cough], which are characterised by protracted apnœa [?], in which cough, with very few or no inspirations, is alone observed, and therefore no whooping tone is heard. *Such attacks are chiefly observed in little children during the first year of life.* The cyanosis rapidly reaches its highest grade; the suffocation is threatening, and may indeed prove fatal, especially when the disease is complicated with diffuse catarrh or broncho-

[1] Diseases of Children, p. 540. [2] Op. cit. p. 173

pneumonia. *Under these circumstances partial spasms (deviations of the eyes, contracture of the fingers, toes, arms, &c.)*, or even *general and fatal convulsions*, may occur during the attack, or immediately afterward, either in consequence of the protracted venous stasis in the brain, or the accumulation of carbonic acid in the blood, which must ensue in the absence of sufficient inspirations."

14. *On so-called Excentric Irritations; Excess of "Natural Stimulants"; Epileptogenous Zones.*

I have said nothing so far of excentric irritations in cases of laryngismus, not being convinced that they have any share in the production of the seizures. I except, of course, that "excess of the natural stimulus" of the respiratory centres which may consist in strong excitation of the vagal ends in the lungs. Dyspepsia may act, if causing flatulence, by interfering with the play of the infant's diaphragm, and thus indirectly help towards increasing the venosity of its blood. In cases of catarrhal laryngitis there may be excentric irritations from the larynx as part causes of the respiratory convulsions; but if so, here again there is an excess of a natural stimulus, although of a different kind from venosity. I should doubt whether dyspepsia (vide *supra*) would by "stomach irritation" cause any kind of fits. What I find hard to believe is, that excentric irritations of any sort, of any part, can act on "the nervous centres" when they are healthy, so as to produce convulsion. I can understand that convulsion may result if the peripheral part irritated is particularly related to (specially represented in) the centre which is compelled to discharge—is its "natural stimulus"—but not then unless the centre is from some other cause already morbidly over-excitable. The part of the periphery (ento or epi) especially united by afferent nerve-fibres to any motor centre, by the intermediation of sensory centres, is, when that centre has become by disease a "convulsive centre," its epileptogenous zone. I can easily understand,

[1] Of course, excitation of very many parts of the body (nose in sneezing, ear in producing cough, sudden application of cold) act on the respiratory centres. I speak of more definite or particular stimuli, and of excessive stimuli.

to illustrate by epileptiform seizures, that if a man have already a "discharging lesion" of part of the so-called "arm centre," a tap on some of the tendons of his hand may *start* a fit. And I do not deny that if a man has epileptic fits (epilepsy proper) with a "stomach warning," indigestion may provoke a fit by producing stomach irritation—the stomach being the epileptogenous zone in that patient. On this topic more later. We next consider cases of respiratory fits, experimentally produced in healthy lower animals, when such excentric irritations need not be invoked, even as part causes. This will be the subject of a future paper.

ON A CASE OF LOCOMOTOR ATAXIA WITH LARYNGEAL CRISES, AND ONE OF PRIMARY SCLEROSIS OF THE COLUMNS OF GOLL, COMPLICATED WITH OPHTHALMOPLEGIA EXTERNA.

BY JAMES ROSS, M.D., LL.D., F.R.C.P.

CASE 1.—W. W., aged 35 years, fishmonger by trade, was admitted to the Manchester Royal Infirmary on Oct. 15th, 1884, under the care of Dr. Ross.

History.—The patient has been much exposed to cold and damp in following his occupation, but he has always had an abundance of good food and warm clothing. His family history is good, and he has himself never suffered from any serious disease up to the date of the present illness. At twenty years of age, the patient was unsteady in his habits, and gave way for two or three years to alcoholic excesses, but since that time he has been a temperate man. In 1870 the patient contracted a sore on the penis, and some weeks afterwards the glands of the groin were swollen, and he subsequently suffered from sore throat and a cutaneous rash. During that time he was under continuous treatment for five or six months; but does not know the nature of the drugs which were prescribed for him. Two years after contracting this sore, the patient married, and eighteen months afterwards his wife suffered from a cutaneous eruption, and her hair fell off. His wife has had one miscarriage, and three still-born and four living children; but of the latter two died from convulsions in infancy, and two are still living and moderately healthy.

The present disease began three years ago, by frequently recurring paroxysms of severe pain in the stomach, which were attended by retching and vomiting of large quantities of bile. The attacks recurred almost every morning at first, and sometimes continued during the whole day, but for the last eighteen months they have only averaged about one in a fortnight.

The patient was also "nervous" in the dark, and six months from the commencement he fell forward one morning into the wash-basin, on covering his face with his hands whilst washing. About this time the patient was sitting one day for some time with his legs crossed, and on attempting to rise he felt a severe tingling in the left foot and leg as far as the knee and found himself quite unable to walk, but he recovered the full use of his limbs in a fortnight.

Coincidently with the appearance of the gastralgic attacks the patient suffered from double vision, and he continued to be troubled with this symptom occasionally for some time; but it was not until fifteen months before admission that he observed the presence of a definite squint, which was accompanied by drooping of the left eyelid. From that time up to a few months ago the left eyelid remained almost completely closed; but during the last few months a considerable improvement has taken place in this respect. About the same time that the eyelid began to droop, the patient noticed a decided failure in the acuteness of his vision, which has progressively increased, until he is now nearly blind, especially on the right side. About two years before admission, the patient began to experience a crowing noise along with inspiration. He suffered almost continuously from this noisy inspiration, but in addition he had paroxysms of difficulty of breathing, each of which lasted several minutes. During these attacks he felt as if he were about to choke, and every inspiration was accompanied by a very loud crow, like that of whooping-cough. The patient states that he was told that the noise which accompanied breathing was always present in a marked degree when he was asleep, and during a residence of a few weeks in the Salford Hospital, some months before he was admitted to the Royal Infirmary, the nurse was in the habit of rousing him several times every night, because his noisy breathing prevented the other patients in the ward from getting to sleep. He suffered occasionally from a girdle sensation on a level with the epigastrium, and had some numbness in his feet; but he appears to have been singularly free from lightning-pains, and up to a few months before he came under observation he had no great difficulty in walking, except in the dark. During the last two or three months his

power of walking rapidly failed, and now he is quite unable to stand. In the early part of his illness the patient observed that he could go a whole day without having any desire to pass water, but on emptying his bladder as a matter of routine at night, he voided a large quantity. This tolerance of the bladder soon gave place to irritability of it, and for some time he has been compelled both to micturate frequently and to obey the call promptly, but, notwithstanding all his precautions, he occasionally dribbles. He suffers much from constipation, and is not always conscious of it when his stools are passed, while he has occasionally soiled the bed.

Present Condition.—The patient lies on his back, and although he can move his legs about freely when laid down, he is quite unable to stand. He is a thin, spare man, and considerably emaciated, but none of the muscles appear to have undergone special atrophy. The patellar-tendon reactions are absent on both sides, ankle-clonus cannot be elicited, and there is no muscular tension or contractures. The patient states that both feet feel numb, and the prick of a pin does not give rise to much pain in the skin below the knee, while several seconds intervene between the prick and the recognition of the pain. Two points have to be separated about eight inches before they are felt as separate over the skin on the outside of the leg. The patient has also a very imperfect appreciation of the position of his legs when his eyes are closed, and the muscular sense, as tested by different weights attached to the legs, is very deficient. Sensation, as tested by touch and pricking, is impaired in both hands; the loss of sensibility being specially marked in the ulnar half of the hand and two and a half fingers. On being asked to touch with closed eyes the point of his nose by the tip of one of his index-fingers he touches wide of the mark, the finger sometimes hitting one of the eyes instead of the nose. The patient fumbles a good deal when buttoning and unbuttoning his clothes, and he experiences considerable difficulty in seizing small objects. Both eyelids are somewhat drooping, but the left is affected to a much greater extent than the right; and the cornea of each eye is drawn somewhat towards the outer canthus, owing to a predominance in the action of the external rectus muscle, and

consequently the patient has a double divergent squint. All the movements of the eyeballs, however, are much restricted, and even outward rotation is not carried out to the normal extent, while the power of rotating the eyeballs upwards and downwards is almost entirely lost. Inward rotation is effected better on the right than on the left side, and indeed the paralysis of all the muscles is less marked on the right than on the left side. The pupils are always moderately dilated; but, as a rule, the right is not quite so large as the left. The pupils fail to contract to the stimulus of light, and they only contract to a slight extent to accommodation. The patient can count figures held up before him with either eye, but he is quite unable to read, having a difficulty in distinguishing single letters. The optic discs are found, on ophthalmoscopic examination, to be in a state of advanced white atrophy. The patient speaks in a peculiar drawling manner—his voice has a slightly nasal quality, and at the end of each sentence the drawing in of the breath is accompanied with a slight noise, like that caused by air drawn through a narrow reed. At night, when the patient is asleep, the noise which accompanies inspiration is said to be much louder than when he is awake; and the nurse states that the other patients near him are frequently disturbed in their rest by it. The patient has to keep a urinal in constant use, and occasionally he has soiled the bed by an unconscious passage of fæces. He was kept five weeks in the Royal Infirmary, during which he was treated with rest in bed, ten-grain doses of iodide of potassium three times a day, and good diet. He was sent to the Convalescent Hospital at Cheadle on Nov. 22nd. Dr. Wansbrough Jones kindly took the following notes for me:—

Jan. 31, 1885.—For the first few weeks after the patient's admission at the Cheadle Convalescent Hospital he sat up a great part of the day, but he was quite unable to stand or walk without assistance. Five weeks after his admission he began to be troubled with severe lightning-pains in his lower extremities, and with attacks of vomiting, which recurred almost daily, and were accompanied by severe lightning and girdle pains. Each attack of vomiting lasted from four to six hours, and the patient was only maintained in tolerable

comfort by having subcutaneous injections of morphia. Every deep inspiration is accompanied by a stridulous noise, and this noise is particularly loud when the patient is asleep; but in addition he suffers from severe suffocative paroxysms, each of which lasts several minutes, while they recur on an average twice a week. During the attack each inspiration is accompanied by a loud crowing, like that of whooping-cough or of laryngismus stridulus. A laryngoscopic examination shows that the vocal cords do not attain the normal excursion during respiration, and that the chink of the glottis is much smaller than usual during inspiration. About ten days ago the patient had an attack of herpes over the outer surface of the right ear, which lasted nearly a week. About a fortnight ago three large boils appeared over the sacrum, and now ulcerated surfaces are left, the largest of them being about the size of a crown-piece. The disorders of the movements of the eyeballs and pupils, and the other symptoms, have remained much the same as they were when the patient was at the Infirmary.

Feb. 20.—The ulcers over the sacrum became rapidly cicatrised, but soon afterwards the patient was found to have a remittent temperature, and fluctuation was felt over the sacrum; the skin was punctured, and a considerable quantity of pus was voided. He is still suffering greatly from lightning-pains, and gastric and laryngeal crises, and has to get progressively larger doses of morphia in order to make life tolerable. The abscess over the sacrum continues to discharge; temperature is of remittent type; the patient appears to lose strength daily, and to be undergoing progressive emaciation; and he was consequently sent back to the Royal Infirmary on March 4, 1885.

March 22.—On being readmitted into the Infirmary, the patient lay in bed, and had to be kept under the influence of morphia. His stools are often passed unconsciously in bed, and his urine dribbles, so that a urinal has to be in constant use. The urine is alkaline, and contains numerous bacteria, pus-cells, and crystals of triple phosphate, and the temperature varies from 97·5° in the morning, to 101° in the evening, while the abscess over the sacrum still continues to

discharge. The lightning-pains, and gastric and laryngeal crises and other symptoms, continue much the same as they were at Cheadle. The patient is discharged on April 8, 1885.

April 27.—At home the patient was attended by Mr. Pilkington, District Surgeon to the Royal Hospital, Salford. Mr. Pilkington informed me that he got gradually weaker from the time of leaving the Infirmary, and he at last died April 24; three days later Mr. Pilkington was kind enough to make a post-mortem examination, and to send me the crura cerebri, pons, medulla oblongata, and spinal cord. These portions of the nervous system I handed over to Dr. Harris, Pathologist to the Royal Infirmary, and, after hardening, he was good enough to cut sections for me. The spinal cord itself was so brittle after hardening, that it was not found possible to make sections of it, but it could be seen with the

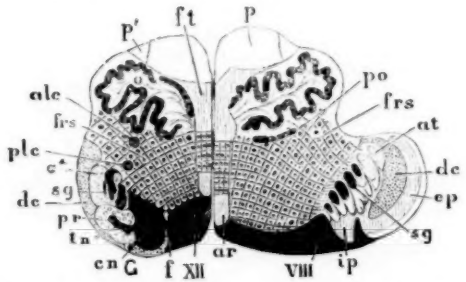

FIG. 1 (modified by Young from Flechsig).—p p,' pyramidal tracts XII-VIII. nuclei of the twelfth and eighth nerves respectively; a r, slender, and p r, cuneate fasciculi respectively; d c, direct cerebellar tracts; a t, ascending roots of the fifth nerves; f, fasciculus rotundus.

naked eye that the whole of the posterior columns, and probably also the direct cerebellar tracts, were diseased throughout their vertical extent. An examination of a transverse section of the medulla oblongata, on a level with the middle third of the olivary body (Fig. A, left half), showed that the sclerosis had extended to the thin layer of white substance which lies over the surface of the cuneate nucleus (Fig. 1, G) and which is the upward extension of the column of Goll, to the layer of white fibres which lies to the outer side of the triangular nucleus (Fig. 1, p r), and which is the upward continuation of

the posterior root-zones, and to the layer of white substance which occupies the postero-lateral surface of the medulla oblongata (Fig. 1, d c) and which represents the direct cerebellar tract. Considerable evidences of disease were also manifest in the ascending root of the fifth nerve (Fig. 1, s g), while the fasciculus rotundus (Fig. 1, f) was almost completely disorganised, this tract being traversed by a relatively large vessel with thickened walls, and its substance studded with nuclei, amidst which no healthy nerve fibres could be discovered. A large number of the root-fibres of the vagus were found atrophied. An examination of sections of the crura cerebri

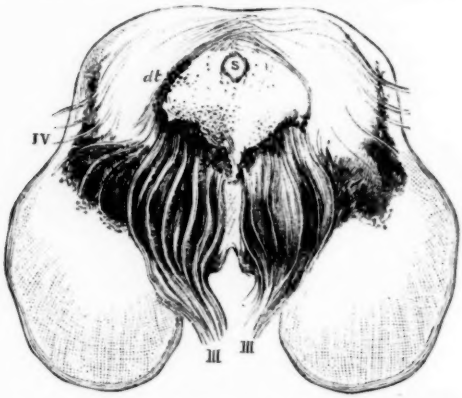

FIG. 2 (after Young).—Section of the crura cerebri. The left lateral half represents the healthy crus, and the right, the crus in the case of W. W.; *d t*, descending roof of the fifth nerve, with fibres from the anterior tubercle of the corpora quadrigemina passing transversely through it; s, aqueduct of Sylvius; III, III, third nerves; IV, fourth nerve.

revealed considerable changes in the nuclei of the third and fourth nerves and neighbouring parts. The grey substance on the floor of the aqueduct of Sylvius was studded with nuclei, and the ganglion cells of the nuclei were deficient in number, and many of those left were small rounded bodies destitute of processes, these changes being more pronounced in the nuclei of the left than those of the right crus. The fibres of the third nerve, when examined with a high power, could be seen as fine hairs in their passage through the tegmentum and crusta,

and the medullary sheath seemed to have disappeared from a large number of the fibres. Scarcely a trace of the bundle of longitudinal fibres which constitutes the descending root of the fifth nerve could be discovered, while the fibres from the anterior tubercle of the corpora quadrigemina which pass through this tract to reach the nucleus of the third nerve were interrupted and atrophied. The annexed diagram (Fig. 2) was kindly drawn for me by my friend Professor Young. The left half of the figure represents a healthy crus, while the right half represents the changes observed in this case. It will be seen that on the right side, as compared with the left, the ganglion cells of the nucleus of the third and fourth nerves are small and few in number, that the third nerve (III) is atrophied, and that the descending root of the fifth nerve (*d t*), with the fibres passing through it from the corpora quadrigemina, has disappeared.

Remarks.—The chief interest in this case centres in the morbid changes found in the cranial end of the central grey tube. In the medulla the principal morbid changes were observed in the white substance of the slender (Fig. 1, G) and cuneate (Fig. 1, p r) fasciculi and the direct cerebellar tracts (Fig. 1, d c), these being respectively the upward continuations of the columns of Goll, the posterior root-zones, and the direct cerebellar tracts of the cord. The fasciculus rotundus (Fig. 1, f) was also found almost completely disorganised on both sides, while minor changes were discovered in the ascending roots (Fig. 1, a t) of the fifth nerves. These bundles are most probably the homologues in the medulla oblongata of the posterior root-zones of the spinal cord, the fasciculus rotundus being associated with the lateral mixed system of nerves consisting of the spinal accessory, vagus, and glosso-pharyngeal nerves, and the ascending root of the fifth being connected, as its name implies, with the great sensory nerve of the head. Changes like those just reported, have already been described by Pierret[1] as occurring in the medulla oblongata in cases of

[1] Pierret, 'Essai sur les symptômes céphaliques du tabes dorsalis,' Paris, 1876, p. 19; et, 'Sur les relations du système vaso-moteur du bulbe avec celui de la moelle épinière chez l'homme et sur les altérations de ces deux systèmes dans les cours du tabes sensitif,' Comptes Rendus, 1882, No. 5, 30 Janv. 1882.

locomotor ataxia, and it is consequently unnecessary for me to make any further remarks upon them in this place. The changes observed in the root-fibres of the vagus in this case corresponds with the cases of tabes with laryngeal crises, reported by Cruveilhier[1] and Jean,[2] in which the roots of the pneumogastric nerves were found atrophied. The most important changes, however, discovered in this case were those found in the crura cerebri. Passing over the morbid alterations of the ganglion cells of the nucleus of the third and fourth nerves, we come at once to speak of those observed in the bundle of white fibres which is named the descending roof of the fifth nerve (Fig. 2, *d t*). This bundle is most probably homologous in its structure and functions with the ascending root of the fifth, which is in its turn, as already stated, homologous with the posterior root-zone of the spinal cord. It is therefore probable that disease of this bundle gives rise to the diplopia and disorders in the movements of the eyeball which so frequently appear in the early stages of tabes, even in the absence of any disease of the ganglion cells of the nuclei of the ocular nerves, although it is highly probable that in the more marked forms of ocular paralysis the ganglion cells are always implicated. It will also be seen from the diagram (Fig. 2) that a small bundle of fibres passes transversely through the descending root of the fifth nerve. These fibres join the anterior tubercles of the corpora quadrigemina with the nuclei of the third and fourth nerves, and it is very probable that some, if not all of them, complete the arcs between afferent fibres in the optic nerves and tracts and efferent fibres in the third nerves, the whole constituting the mechanism which regulates the light-reflex movements of the pupils. The annexed diagram (Fig. 3), borrowed from Erb, shows the nervous mechanism which regulates the movements of the pupil. The light-reflex contraction of the pupil is regulated by the arc (G B C H), passing from the retina (F) to the sphincter of the pupil (E). A lesion in the afferent part of the tract will arrest the reflex contraction of the pupil, but the loss

[1] Cruveilhier, 'Anatomie Pathologique,' Paris, 1835–42; Liv. xxxii. p. 19.

[2] Jean, 'Ataxie locomotrice progressive. Troubles ataxiques du côté du larynx et du pharynx,' Gaz. Hebdom. Journ. ii. 1869, p. 121.

of this reflex will be accompanied by complete blindness; and disease of the efferent fibres (H) will also arrest the reflex, but this loss will be accompanied by dilatation of the pupil and loss of contraction of the pupil to accommodation, because the conducting path from the cortex of the brain at (A) will be injured. But disease of the fibres (Δ) which connect the anterior tubercles of the corpora quadrigemina (B) with the nucleus of the third nerve (C) will arrest the light-reflex, and the loss of the reaction will not be accompanied either by blindness,

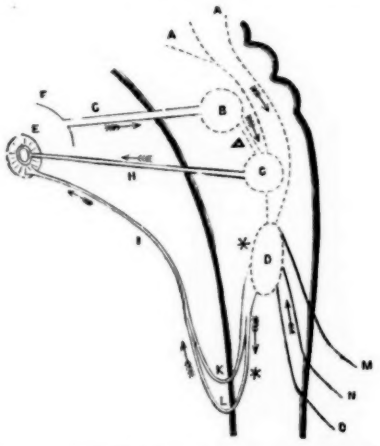

FIG. 3 (after Erb).—A A, psychical impression; B, anterior tubercle of corpora quadrigemina; C, oculo-motor centre; D, dilator centre (spinal); E, iris; F, retina; G, optic nerve; H, oculo-motor (sphincter); I, sympathetic (dilator); K, L, anterior roots; M N O, posterior roots; Δ, seat of lesion causing reflex pupillary immobility; *, probable seat of lesion causing myosis.

paralysis of the sphincter of the iris, or loss of contraction to accommodation. In this case the patient was partially blind, and the sphincters of the pupils were partially paralysed, but neither the blindness nor the paralysis was complete, and consequently the loss of the pupillary light-reflex must have been caused by disease of the fibres connecting the corpora quadrigemina with the nucleus of the third nerve. When,

[1] For a discussion of the present state of our knowledge of the lighter reflex of the pupil, see Dodds, W. J., "On some Central Affections of Vision;" 'BRAIN,' Part XXXI., Oct. 1885, p. 345.

therefore, we meet with the Argyll-Robertson pupil, it may be inferred that the fibres which connect the anterior tubercle of the corpora quadrigemina and the nucleus of the third nerve are diseased, most probably on both sides; and in locomotor ataxia these fibres are most likely to become secondarily diseased in their passage through the longitudinal fibres which constitute the descending roots of the fifth nerves. It will thus be seen that loss of the light-reflex contraction of the pupil is one of the most surely localising symptoms of disease of the nervous system which we know.

The notes of the following case were taken by my Clinical clerk, Mr. Sidebotham:—

Case II.—D. G., ætat. 35 years, a collier, was admitted to the Manchester Royal Infirmary on April 15th, 1885, under the care of Dr. Ross.

History.—The patient has worked in a coal-pit from a very early age, and has been much exposed to cold and damp, but always lived comfortably. He has indulged pretty freely in alcohol, but he says that he never drank to excess. When a young man he had a sore on the penis, on the glands of which a cicatrix is still to be seen; but he does not seem to have suffered from sore throat or cutaneous eruptions. He has been married twelve years, and his wife had a miscarriage soon after their marriage, but she has had no living children. The patient enjoyed remarkably good health up to the date of his present illness. About three years ago the patient felt for the first time a weakness about the knees, and this gradually increased so much, that at the end of a few months he was compelled to leave off work, and he has never been able to resume it. Until lately he has not suffered from any of the paræsthesiæ, and other sensory disorders which are so common in the first stage of locomotor ataxia, and it is only three weeks ago that he felt for the first time a slight girdle sensation passing round the lower part of the chest. He has not apparently suffered from any lightning-pains.

Present Condition.—The patient is a strong, muscular man, having a dull and sleepy expression, chiefly owing to well-marked double ptosis. The eyeballs are almost completely immovable in every direction; but the pupils, which are some-

what dilated, contract readily both to light and to accommodation. The patient has considerable difficulty in recognising colours, and his vision is somewhat impaired, while an ophthalmoscopic examination reveals a considerable degree of pallor of both optic discs. The patient speaks in an indistinct and drawling manner, and he has to balance his words like a man in the first stage of intoxication. He states that his articulation was at one time very distinct and clear, and that the present defect is of recent date. There is some degree of anæsthesia of the skin of the lower extremities, as high up in the front as the junction of the upper and middle thirds of the legs, and behind as the popliteal spaces. The patient knows the position of his legs accurately when his eyes are closed. The patellar tendon-reactions are completely absent, and no ankle-clonus can be elicited. When his feet are placed side by side along their inner edges, the patient sways a little from side to side on closing his eyes, but with a little effort he can maintain himself erect. In walking, his gait is somewhat straddling, uncertain, and staggering, but not like that of locomotor ataxia. The patient will not walk much without the support of a stick, as otherwise he would, he says, stumble or fall. In walking, his feet are kept widely apart, and the advancing foot is raised to an abnormal degree from the ground; but it is not brought down with a thump, and the heel does not strike the ground first, as in ataxia. The body also rocks a little from side to side in walking, but it is more like the reel of cerebellar disease than the balancing of the body in tabes. The patient says that his knees are liable to give way suddenly, as if his legs were paralysed; and if he were not to exercise great care in supporting himself with the stick, he would often fall to the ground. He was sent to the Cheadle Convalescent Hospital on May 18th, and for the following particulars of his case I am indebted to Dr. Wansbrough Jones.

The notes taken by Dr. Jones confirm on the whole those taken by Mr. Sidebotham at the Infirmary. The only new symptom observed was that during the last few weeks of his life he began to complain of lightning-pains in the lower extremities, but they never attained to any great severity. Notwithstanding his sad condition, the patient was very lively and fond of

joking, and had the reputation amongst the patients of being a "romancer;" but there is nothing to show that he ever manifested any of the "grand ideas" which might be considered to indicate general paralysis. On July 13th the patient had just taken his medicine—ten grains of iodide of potassium—and immediately went to the smoke-room, where he began to joke with some of the other patients. In a few minutes afterwards he suddenly became insensible, and was carried to bed. After a few minutes he recovered so far as to ask for a drink, but was soon seized with a second apoplectiform attack, from which he again made a partial recovery, to be almost immediately seized with a third attack, in which he died, the time from the commencement of these attacks till death not being more than ten minutes.

The autopsy was conducted by Mr. Corbett at noon, on July 15th, in the presence of Dr. Wansbrough Jones and myself. The brain was found somewhat congested, and about an ounce of fluid escaped during removal; but no other evidence of disease was discovered, either on the surface of the organ, or after the most careful slicing. The spinal cord, medulla oblongata, pons, and crura cerebri, were reserved for microscopical examination. The heart, large blood-vessels, lungs, kidneys, and all the other internal organs, were found healthy. On examining sections of the spinal cord, some stained by carmine and others by Weigert's method, kindly prepared for me by Dr. Harris, it was found that the columns of Goll were in a state of sclerosis (Fig. 4), throughout their whole longitudinal extent in the cord, but no decided morbid changes could be discovered in the upward continuation of these tracts in the medulla oblongata (Fig. 1, G). But although the disease was most marked in these columns, it was not strictly limited to them in the inferior portion of the cord. On a level with the fourth pair of sacral nerves (Fig. 4. S_4), a level at which, according to Flechsig, the columns of Goll are not represented, the diseased patch occupies a small triangular area, the apex of which hardly reaches as far forward as the middle of the posterior median fissure. The base of the triangle is directed to the posterior surface of the cord; but on this surface the disease extends laterally, and passes along

the outer surface of the internal bands of the posterior columns—Burdach's columns—as far as the posterior roots. On a level with a fifth pair of lumbar nerves (Fig. 4, L_5) the apex of the medium triangular diseased area reaches further forwards than in the last section, and on attaining to the level of the third pair (Fig. 4. L_3) it reaches quite up to the posterior commissure; while on the posterior surface of the

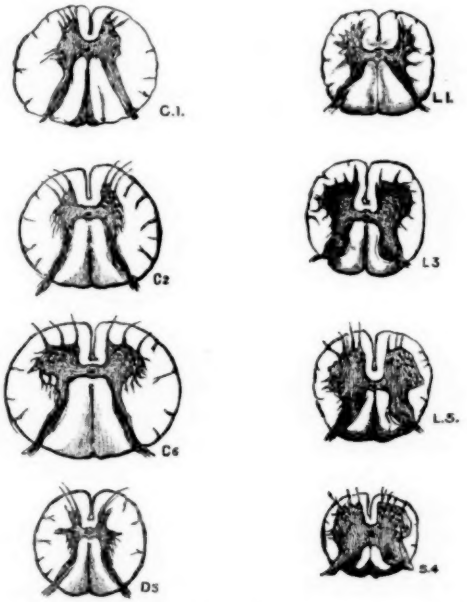

FIG. 4 (after Young).—Sections of the spinal cord from the case of D. G., on a level with the 4th sacral, 5th, 3rd, and 1st lumbar, 5th dorsal, and 6th, 2nd, and 1st cervical pairs of nerves respectively. The shaded portions in the posterior columns represent the transverse distribution of the sclerosis.

cord at both levels the disease extends laterally up to the posterior roots. On a level with the first pair of lumbar nerves the disease has the same kind of distribution as on the level of the third pair, except that at the former of the two levels it extends laterally to some slight extent along the posterior commissure, while a small patch of healthy fibres are

observed on each side of the middle third of the posterior median fissure. On a level with the first, second, and third lumbar nerves the posterior roots and posterior horns appear to be implicated in some slight degree in the disease. An examination of sections at different levels in the dorsal region shows that on ascending from the first lumbar pair of nerves the diseased area gradually recedes, so that on a level with the fifth pair of dorsal nerves (Fig 4, D_5) the apex of the median triangle does not quite reach up to the posterior commissure, and the lateral extensions on the posterior surface fall short of the posterior roots, although still invading to a slight extent the external bands of the posterior columns. In the upper dorsal and inferior cervical regions (Fig. 4, C_8) of the cord the disease becomes strictly limited up to the columns of Goll, while in the upper cervical region (Fig. 4, C_2 and C_1) only a small portion of these columns is implicated; but the annexed figures render unnecessary a further description of the distribution of the disease at these levels.

In the medulla oblongata the fasciculus rotundus was probably the subject of a slight degree of sclerosis on both sides, but the ascending roots of the fifth, and the direct cerebellar tracts, were found healthy. In the crura cerebri, the grey matter on the floor of the aqueduct of Sylvius stained deeply with carmine, and when examined microscopically, the tissue at this point was thickly studded with nuclei, while many of the ganglion cells were found to be small rounded masses, destitute of processes. Many of the fibres of the third nerves were extremely fine, and apparently destitute of medullary sheaths. The descending root of the fifth nerve, and the fibres which pass transversely through it, were very distinct, and free from any trace of disease on one side; but on the other side some of the longitudinal fibres had apparently undergone sclerosis, as evinced by a higher staining with carmine, and the absence of the cut ends of the fibres. The diseased fibres were, however, comparatively few in number, and the transverse fibres coming from the corpora quadrigemina were not apparently affected.

Remarks.—The fact, that the disease had extended to the external bands up the posterior columns, renders it doubtful

whether this case ought to be described as a primary sclerosis up the columns of Goll; but as it is probable that the sclerosis had not been quite circumscribed in Goll's tracts in any of the other reported cases of this affection, we see no valid reason for refusing to place the case just described under this category. Passing over the cases recorded by Dr. Castel[1] and Gowers,[2] as not presenting any definite symptoms during life which could be referred to the disease found in Goll's columns, we come to consider the classical case reported by Pierret.[3] The drawings which illustrate the report of this case show that the disease of the median columns must have encroached to some extent on the external bands on a level with the twelfth dorsal nerves, much in the same way as it did in the middle dorsal region in my own case. No special mention is made by Pierret of the distribution of the sclerosis on a level with the first and second lumbar pairs of nerves, but a little above the middle of the lumbar enlargement a patch of disease was noticed on each side of, but somewhat removed from, the posterior median fissure, and, if we take Flechsig's diagram as our guide, these patches must have been situated, not in Goll's, but in Burdach's columns. In a case of primary sclerosis of the columns of Goll reported by Kahler and Pick,[4] of which we regret to say we have only seen a short abstract, it is stated that the disease was "tolerably accurately" limited to Goll's columns, and it may therefore be inferred that the morbid changes had encroached to some extent on Burdach's columns. In a case of this affection reported by Camuset,[5] the description shows that the disease had much the same kind of distribution as was observed in my own case, and it is expressly stated that in the lumbar region the morbid changes had extended so as nearly to reach the posterior roots.

[1] Du Castel, "Sclérose primitive des cordons de Goll;" 'Gaz. Méd. de Paris,' 1874, No. 3.

[2] Gowers, 'Lancet,' vol. ii. 1879, p. 876

[3] Pierret, "Notes sur un cas de sclérose primitive du faisceau médian des cordons postérieurs;" 'Arch. de Physiologie,' 1873, p. 74.

[4] Kahler und Pick, "Zur Lehre von der Fasersystemen in den Hintersträngen des Rückenmarks;" 'Prager Zeitschrift für Heilkunde,' Bd. ii. 1881, p. 317. Abstr. Virchow und Hirsch 'Jahresb.,' Bd. ii. 1881, p. 93.

[5] Camuset (L.) "Sclérose primitive des cordons de Goll;" 'Le Progrès Médical,' tome xii. 1884, p. 1008.

A study of the cases collected and analysed by Strümpell[1] and Adamkiewicz[2] shows that the portion of Burdach's columns which are usually spared to the last in cases of ordinary locomotor ataxia—viz. the posterior third and the anterior part adjoining the posterior commissure—were the parts which alone were attacked in the case just reported, thus showing that this case differs greatly in its morbid anatomy from ordinary cases of posterior sclerosis. The presence of ophthalmoplegia externa and the absence of the patellar-tendon reaction in my case, however, show that there is a close relationship between primary sclerosis of the columns of Goll and genuine locomotor ataxia; and it is very probable that had my patient lived longer, the disease would have implicated the external bands of the posterior columns to a much greater extent than it had done, and the case would then have been regarded as an example of the ordinary form of *tabes dorsalis*.

As regards symptoms, Pierret's case suffered from numbness and lightning-pains in the limbs, and girdle-sensations passing round the body; and in Kahler and Pick's case there were first violent lumbar pains and tearing and drawing pains in the legs; but in Camuset's case there was a marked absence of lightning-pains or other sensory disturbance; while in my case there was some anæsthesia of the legs, but the lightning-pains appeared for the first time only a few weeks before the patient died.

The gait of the patients whose cases are reported by Pierret and Camuset was not unlike that of my patient; in all the cases walking was impossible without the aid of some support—a crutch or a stick, and the walk consisted of an uncertain stagger, rather than a genuine ataxia. Loss of the patellar-tendon reactions was not known as a symptom at the date of Pierret's observations, and the state of these reactions is not mentioned in the abstract of Kahler and Pick's paper, or by Camuset; but the reactions were absent in my case. It is

[1] Strümpell (A.), 'Archiv für Psychiatrie,' Bd. x. 1880, p. 676; Bd. xi. 1881, p. 27; and Bd. xii. 1882, p. 723.

[2] Adamkiewicz, "Die anatomischen Processe der Tabes dorsualis;" 'Aus dem XC Bande der Sitzb. der K. Akad. der Wissensch.' III. Abth.,' October Heft. Jahrg. 1884.

probable that loss of these reactions was caused by the slight lateral extensions of the disease to the posterior root-zones at the posterior surface of the cord, rather than to disease of the columns of Goll themselves. The presence of swaying movements on the eyes being closed in this case, tends to show that this symptom is caused in locomotor ataxia by implication of the columns of Goll. This conclusion is, however, rendered doubtful by the fact, that the difficulty of maintaining the erect posture was not increased on closing the eyes in Camuset's case. The activity of the light-reflex of the pupil during life in this case, and the absence of any evidence of disease of the transverse fibres which pass through the descending roots of the fifth nerves, furnish, to some extent at least, the negative side of the proof of the theory, that loss of the light-reflex is caused by disease of these fibres. The symptoms which immediately preceded death in this case, show, like many other reported cases, that a patient may die suddenly with all the phenomena which attend a massive hæmorrhage in the brain, without anything being found after death, either in the brain or other organs, to account for the fatal issue. It is worthy of note that in the case reported by Camuset the disease proved fatal by an acute cerebral seizure. The patient, who had always suffered from a slight feebleness of the mental faculties, was at last seized with a severe attack of acute mania, which proved fatal at the end of twelve days. In the case reported by Kahler and Pick, the patient died from incontinence of urine, cystitis, bed-sores, and septic fever, this being the most usual mode of termination of all chronic affections of the inferior part of the spinal cord. It is difficult to account for the presence of these symptoms unless the disease had extended beyond the area of Goll's columns in the upper lumbar region on a level with the automatic centres of the bladder and rectum. In my case the whole area of Goll's columns was diseased in the dorsal and lumbar regions of the cord, yet the patient did not suffer from any disorder of the functions of the bladder, or from bed-sores.

ON THE RELATION BETWEEN THE POSTERIOR COLUMNS OF THE SPINAL CORD AND THE EXCITO-MOTOR AREA OF THE CORTEX, WITH ESPECIAL REFERENCE TO PROF. SCHIFF'S VIEWS ON THE SUBJECT.

BY VICTOR HORSLEY, B.S., F.R.C.S.,

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THE problem which most attracts the attention of all classes of neurologists, whether clinical, experimental, or anatomical, is the identification of function with certain spots in the brain, and discovery of the channels of communication which unite the central nervous system with a harmonious whole. It is the discussion of the relation between the afferent paths in the spinal cord and the motor cortex which concerns us in this paper, renewed attention having been lately drawn to the subject by the recently published experiments of Prof. Schiff. Briefly we may state the case, with a slight preamble, as follows. Every one admits the necessity of regarding the whole central nervous system as being built up on the plan of (1) an afferent channel leading to (2) a sensory centre, which is in close communication with (3) a motor centre from which issues (4) an efferent channel.

Now the bare existence of these constructive factors being granted, it is only their anatomical arrangement which is hotly disputed. There may be said to be three chief views on this subject.

A. The "Separatist" View.—This view places the sensory (perceptive) centres in a distinct, though neighbouring, part of the cortex from the motor centres which are situated in the excitable area of the same. The posterior columns or afferent channels, therefore, are in direct communication with

the former of these centres, and only reach the latter (the motor) indirectly by the commissural connections between the centres.

B. The "Fusion" View.—A favourite mode of regarding the subject on the Continent appears to be one in which the sensory (at least that for tactile sensibility) centre is fused with the motor centre. This view, it is to be supposed, carries with it the assumption, that the posterior columns of the spinal cord are in direct communication with the motor cortex.

C. Prof. Schiff's View.—Prof. Schiff states that he has traced (physiologically) the afferent fibres (of the posterior columns of the cord) directly to the excitable portion of the cortex, which he therefore regards as being the sensory perceptive centre for tactile sensibility. From this sensory perceptive area he believes that fibres go down to the motor centres, which are situated he knows not where.

To recapitulate; it is clear that, according to the first view, (2) and (3) are situated in different parts of the cortex, (3) being identical with the excitable area. In the second view, (2) and (3) are fused together in the excitable area; while finally, according to Schiff, (2) is identical with the excitable area, and (3) is somewhere else.

This much being clear, it is now my intention to detail at length Prof. Schiff's mode of reasoning and experimentation, which has led him to the above conclusion. I shall then point out the fallacies in the same, and finally describe fully the experiments I made last autumn to investigate this point. We owe the unusual opportunity of learning much of Prof. Schiff's work to the scientific zeal and energy of Dr. W. R. Huggard, who communicated to the 'Lancet' of Aug. 1, 1885, a paper describing the above-mentioned work, and entitled, "Professor Schiff's Experiments on the Excitable Area of the Cerebral Cortex." In that paper he states pretty fully the way in which Schiff looks at the subject, and his method of investigating the same. Before entering on the practical side of the matter in dispute, I shall criticise the line of reasoning and the points of explanatory meaning, which occur in Dr. Huggard's report.

Such examinatory criticism must necessarily be somewhat disjointed, as the tale which is told in the above-mentioned

paper is, in the nature of things, a general essay on the work of the distinguished Genevese Professor. Possibly, however, very little difficulty will be met with in picking up the general thread of the argument. We may note at the outset an enunciation (p. 8 of separately published copy of Dr. Huggard's paper[1]) of the function of the excitable area of the cortex which is intended to be descriptive of the action of a sensory perceptive centre, but which, as a matter of general understanding, is, more properly speaking, a perfect explanation of the function of a motor centre. The passage runs thus: "The grey matter of the excitable area transmutes sensibility into a liberating (probably voluntary) current." The usual interpretation which is put on such a term as "sensibility" is the appreciation by a sensory perceptive centre of message conveyed thereto by an afferent channel, for consciousness is connoted. Hence Prof. Schiff's real position is that the excitable area is a portion of brain which transmutes the energy received from a sensory perceptive centre into a motor impulse ("*voluntary current*") and that is one which all upholders of the first or separatist view (A. before described) will cordially agree to.

It seems to me very necessary to dwell upon these apparently trivial differences in modes of expression, for it is just ambiguity in the meaning of terms which of itself creates diversity of opinion where very often agreement really exists. To pursue this point a little further, we find that Prof. Schiff (and following him Dr. Huggard too) is disposed to consider that certain apparently paretic (I say apparently, merely for the sake of argument, as I shall show directly the condition is one of real paralysis) conditions are capable of being referred to as ataxia. Perhaps it will be better to illustrate Prof. Schiff's position by the statement of the experimental result on which he bases this opinion. He has found that removal of the sigmoid gyrus, on the one hand, and the effects (after four or five days have elapsed) of cutting the posterior column of the cord on the other, are both productive of similar symptoms, viz. paresis (voluntary paralysis, really),

[1] Wherever paging of Dr. Huggard's paper is mentioned hereafter, it is to be understood as belonging to the Separate publication.

and that this apparent paresis disappears when the animal runs about on a rough surface. The condition following these operations he calls ataxia.

The adoption of this term to express the condition which results from destruction of the sigmoid gyrus might perhaps be justified, if the exactly similar symptom was evoked by section of the posterior columns of the cord only. Prof. Schiff, of course, considers this latter point is established by his experiments; but I shall show directly that (to use Dr. Huggard's words) "they do not warrant the inference," and in fact that "an essential factor has been overlooked." To proceed with the examination of this so-called ataxic condition, it will be well worth while to describe in greater detail the effects of removal of the sigmoid gyrus in the dog, and the more especially since the hiatuses in Prof. Schiff's description, or perhaps I should more correctly say Dr. Huggard's report, happen to include the omission of some of the most characteristic features. Thus the position of the paresed limbs is very constant and striking. The animal stands resting mainly on the limbs of the opposite side, and with the toes of the paralysed side doubled up, so that the dog walks really on the dorsum of the manus and pes respectively. Precisely the same condition sometimes follows section of the posterior columns of the cord, for the simple reason (see p. 5) that the pyramidal tracts frequently degenerate at the seat of operation, and further, exactly the same condition follows section of the principal nerve-trunks in the limb itself. Next, if thorough examination be made of such an animal, it will be found that there is complete paralysis of voluntary movement, the animal is unable to purposely extend the toes and wrist, &c. It should really be scarcely necessary at the present time to point out again the enormous difference in the proportion of cortical representation of the peripheral muscular segments in the dog as compared with other animals, but from internal evidence in the above-mentioned report it does not appear that this fact is fully appreciated yet. The amount of differentiation exhibited by the various so-called centres is extremely small in the dog. Even the extensor muscles of the paw and digits, the representation of which

may be considered as the acme of cortical evolution in the dog, can very rarely be called into solitary action by stimulating the cortex even with minimal currents. And just as local differentiation in the extreme point of cortical evolution only crudely exists, so also the extent of the excitable area for the limb movements is extremely small compared with the rest of the brain, hence it is clear that in such an animal the very primitive phenomena of equilibration and progression are most unlikely to be disturbed by removal of the mechanism of those volitionary impulses which occasionally mollify them. To sum up this point, it is clear that whatever part of the volitional motor tract be injured, whether it be the nerves in the limb (provided, of course, all the motor nerves in the limb are not cut, but only those supplying the two main distal segments), the pyramidal tracts in the cord, the internal capsule or the cortex itself, the result is the same as might have been expected. It only remains now to take note of the two points on which most stress is laid by Prof. Schiff, namely, first his assertion, that removal of the excitable area in the dog is followed by loss of tactile sensibility on the opposite side; and secondly, his "crowning evidence" (pp. 6, 7), which consists of the following observation: "In dogs whose posterior columns were cut in the cervical region, if, after the lapse of about four days, the so-called motor region is laid bare, it is found on both sides to be no longer excitable. If one posterior column only is cut, the opposite side of the brain only is inexcitable. If the posterior columns or posterior roots (?) are cut in the lumbar region, irritation of the cortex still produces movements of the anterior limbs, but not of the posterior."

Now as regards the first of these two points, viz. the asserted disappearance of tactile sensibility after removal of the opposite sigmoid gyrus, I cannot speak from personal observation on dogs, and therefore simply refer the reader to Professor Ferrier's 'Functions of the Brain,' pp. 216–217, where this question and that of ataxia too, is sufficiently discussed. We may therefore put aside the first point and take up the second, viz. his attempted demonstration of the direct extension of the posterior columns of the cord to the sigmoid gyrus

of the opposite side. His method of experimentation, so far as can be made out from the necessarily short description given in the before-mentioned paper, consists simply of exposing and dividing one posterior column, say the left; then preserving the animal for a period of not less than five days; then exposing the opposite sigmoid gyrus and stimulating it with the ordinary induced current, and observing the absence of the usual movements of the left limbs. The section of the posterior column was usually made opposite, about the fourth cervical nerve, and the motor centres for the fore-limbs of the same side tested as described.

The whole gist of this experiment, as ingeniously designed by Prof. Schiff, lies in the provision of the interval of five days between the section of the cord and the testing of the opposite motor gyrus. For he considered that the consequential ascending degeneration of the posterior column would, in that space of time, reach the opposite cortex and cause degeneration of the grey matter there, which grey matter, we have already seen, he believes to be the perceptive centre of tactile sensibility.

My criticism of this experiment is that it does not justify the conclusions drawn from it by Prof. Schiff until microscopic examination has shown that (1) there is such degeneration of the sigmoid gyrus, and (2) that there are no further changes in the spinal cord than the lesion and degeneration of the posterior column. Feeling the extreme importance of deciding the question thus raised by Prof. Schiff, I determined to repeat his experiment, directing my special attention to these two points; and it will be seen from the results I publish below, that in the first place no such degeneration of the sigmoid gyrus can be demonstrated; and in the second place that Prof. Schiff's "crowning evidence" in support of his position is entirely nullified by the fact, that the section of the posterior column of the cord sets up local degenerative changes in the motor tracts of the cord.[1]

I shall now proceed to expand this latter statement, by

[1] English neurologists at any rate will be interested to learn (vide 'Lancet' of the 20th of February, 1886) that Prof. Schiff is about to combat the general opinion that the crossed lateral tract is motor.

detailing the way in which I have repeated Prof. Schiff's experiment and the results I have obtained.

The animal, a dog, being very deeply[1] narcotised with ether, an incision was made directly over the fourth cervical spine, which being removed, a trephine hole was made through the laminæ of the fourth cervical vertebra. (As a rule the fourth was chosen, but in some of the experiments the spinal canal was opened through the fifth.) The dura mater was then incised by a little transverse cut, and the posterior surface of the spinal cord exposed. After the flow of cerebro-spinal fluid had stopped, the left posterior column of the cord was carefully divided by a small puncturing incision made with a sharp-pointed tenotomy knife. The wound was then closed completely, full antiseptic precautions being taken. On this latter point it is necessary to digress a little. I have performed seven experiments; in two of these the antiseptics failed, owing to the fact, that the animals were so deeply narcotised as to cause cessation of respiration, &c. This necessitated artificial respiration, &c., the spray being discontinued and the windows opened, hence the contamination of the wound. In the remaining five the hole in the spine and dura mater was found by me to be completely plugged, with a dense fibrous plaque of cicatricial tissue, and the soft parts completely united; in two instances the skin being bulged by a collection beneath it of wound serum. In the two instances in which the antiseptics failed, the animals of course suffered from septic meningitis; the subdural space in one being distended with pus, while in the other the arachnoid as far down as the middle of the dorsal region was raised from the cord by pent-up sero-pus. The pus of the wound in each of these two cases was fetid, having a sickly odour, and containing numerous putrefactive bacilli. The advent of this septic condition had only this influence on the experiment, viz. that it made Prof. Schiff's result come out very markedly, the reason of which is obvious.

To return to the method of experimentation.[2] With the

[1] On account of the exquisite sensibility of the dura mater.

[2] I desire here to express my indebtedness to my friend Dr. Beevor for kindly assisting me in the record of several of the following observations.

two exceptions given above, the animals did not appear inconvenienced, except by the motor paresis, &c. During the five days' interval between the completion of the above-described operation and the exploration with induced current of the two cortices, careful examination was made to find whether there was any motor or sensory paresis to be discovered in the left fore-limb. The testing of the former was of course designed so as to make it clear whether the disturbance of voluntary movement was such as to cause incapability of performing *purposive* acts. As regards sensation, the testing of tactile sensibility was performed in three different ways. In the first place the skin was touched lightly and painlessly by means of a pin; in the second place, I used the spring clip referred to by Prof. Schiff; lastly, I devised as a specially sensitive test the injection of water on to the part (the nerve reaction of which may have been hampered), the sensibility of which was in question. At the outset, I think it very desirable that it should clearly be understood by those who have to estimate the value of original research in this direction, how very difficult it is to correctly gauge the appreciation by one of the lower animals, and especially a dog, of tactile impressions. It is not scientific to draw hard-and-fast lines of distinction between the sensory appreciation of touch, pain, temperature, &c., although of course there is actual difference between the perception of these various physical conditions; in short, since we are usually dealing with combinations of any two or more stimuli —stimulation, as a rule, is compounded of several factors—it would seem only natural that the paths conveying such stimuli are not likely to be very highly differentiated. This non-differentiation is of course simple enough to understand as an outcome of such confusion of sensory function, but it must not be left out of mind when we come to consider the appreciation of such different sensory stimuli as those I have already described. I will now enumerate the facts which I observed in my own experiments bearing on this and the foregoing point.

As I have found the affection of sensation to be coupled with the affection of movement (*i.e.* voluntary paralysis, Schiff's

ataxia) in a very suggestive way they may, with advantage, be considered together; and as the motor paresis is by far the easier to recognise, and to explain in accordance with the well-understood function of certain tracts in the spinal cord, I shall take that first.

Paresis.—Of the whole number seven of experiments, the question as to whether the condition which I have shown to be perfectly typical of paralysis of voluntary command of the distal segments of the fore-limb existed or not, could only be determined in six, as the first was complicated too much by septic meningitis. Of the remaining six, this paresis was present in three and absent in the rest, although in every case the complete division of the posterior column had been perfectly effected.

Here, then, is a very definite result, which of itself would be quite sufficient to negative the view that the paresis was a form of ataxia; for though the channels of afferent impressions (according to Schiff) had been destroyed in three cases, there was no "ataxy" produced at all. It is clear therefore that the paresis must be due to damage done to a motor tract lying in proximity to the area of section. Now the two well-known divisions of the pyramidal tract, the direct and crossed portions, are both in proximity to the area divided; for while the former, situate in the anterior column, is only separated by the commissures from the posterior column, the crossed lateral tract is still nearer, being only divided from the latter by the slender posterior cornu of grey matter. In every case one or both of these divisions of the motor tract was degenerated (as will be explained directly), in very varying degree. The cases in which no paresis was observable were those in which the degeneration was very slightly marked; while in those instances where it was severe, the paresis was very definite. This fact of "cortical" *i.e.* voluntary paralysis following a localised patch of acute (*vide* infra) degeneration in the pyramidal tract, will not of course be surprising to any one (save those who question the motor function of the pyramidal tract, *vide* note, p. 47); but connected with this observation of the formation of a local block in the motor tract, as a consequence of the division of the posterior column, is the question of the

degree and causation of the tactile anæsthesia referred to by Prof. Schiff.

The exact relations, *i.e.* corpuscle and ganglion-cell connections of the fibres of the posterior columns of the cord, are not yet accurately known, all, in fact, that we do know of their function being derived from experiments (artificial and natural) on the effects which follow their damage. Certain it is from such data that if one posterior column be divided, ascending degeneration can be traced from the point injured as high as the bulb. Whether all the fibres in the column are thus disorganised as far as the top of the spinal cord, is another matter, and one which I cannot enter on now. Granting, then, that the fibres of the posterior column are afferent as to the nature of their function, I consider that the results of two of the experiments (viz. III. and IV.) in which there was no appearance of disturbance of tactile sensibility, although the posterior column was most completely divided, and the ascending degeneration very well marked, throw grave doubt on the idea that the posterior columns convey afferent tactile impressions. In further confirmation of this idea, which, resting as it does on two experiments only, is certainly open to the charge of being too slightly founded, I wish to note fully the instances, also two in number (Experiments II. and VI.), where this loss of tactile sensibility was well marked. In these cases (II. and VI.) there was undoubtedly tactile anæsthesia; but there was in addition very definite motor paresis, and of course, as explained above, distinct degeneration in the *lateral* column. Now this couple of cases, II. and VI., presents the exact converse of the condition seen in III. and IV., where the affection of the motor tract was very slight indeed. Hence we are brought to this juncture: Is the loss of tactile sensibility due to degeneration attacking the direct cerebellar tract in the most posterior portion of the lateral column, or to a similar damage of other afferent fibres in the lateral column? It is, of course, quite superfluous to discuss the existence and function of the direct cerebellar tract, but it is worth while to pause for a moment, and to remember that the exceedingly careful experiments of Mierscher demonstrated (by accurate

measurement of the variations of blood-pressure) that afferent fibres of so-called vaso-motor influence travelled up to the encephalon in the lateral column of the spinal cord. Degeneration, we have just seen, was present in the lateral columns in my experiment when tactile anæsthesia was noted, and again this latter symptom was absent in the cases where the degeneration was scarcely visible. So here we have the crucial test. I cannot help feeling, therefore, that the question as to the path in the spinal cord of the impressions which arouse tactile sensibility must remain undecided.[1]

To sum up. After the above described operation on the spinal cord, *paresis* and *tactile anæsthesia* were only present in those instances where degeneration had attacked the antero-lateral columns.

I will now take up the last division of my subject, and present the results of stimulation of the cortex of the side opposite to the lesion of the posterior column in the spinal cord.

As however the whole seeming abnormal deficiency of function in the excitable area is most obviously due to the degeneration I have referred to, situated in the pyramidal tract opposite the seat of operation and not, as Prof. Schiff would have us believe, in the excitable area, I think it will be best to describe the state of the spinal cord in each of my experiments.

The spinal cord operated on as above was examined in the the following way. After the cortex on each side had been thoroughly explored (*vide* infra) the cord was rapidly exposed (the animal being deeply etherised), and then being still living, the contrast between the dead white of the degenerated ascending posterior column which had been divided, as compared with the almost semi-translucent appearance of the normal one, was very striking.

The cord was then removed and hardened in a 2 per cent. solution of bichromate of ammonia, to which $\frac{1}{3}$ volume of

[1] Not wishing to stretch my point beyond what some might consider justifiable limits, I have purposely called the ascending fibres, described by Miescher as of "so-called vaso-motor influence," but it will be remembered that they were channels of conduction for afferent impressions produced by stimulating the great sciatic nerve.

methylated spirit was added at the end of a fortnight. Two portions of the cord were then taken, viz. (1) the seat of operation with the neighbouring 6mm. above and below the point cut, and (2) a small piece opposite the 1st or 2nd cervical nerve; these were soaked in water, then in gum, and finally cut after freezing. One half of the sections were then stained with aniline blue-black, the other with logwood, and finally mounted in oil of cloves and Canada balsam. This method of preparing the sections, although rapid, had the disadvantage of allowing the more friable grey matter of the posterior cornu, forming, as it necessarily does, the outer boundary of the wound in the spinal-cord to partly break away in some cases during the preparation of some of the sections, as is represented in the accompanying outlines of the various sections. (See pp. 56, 57.) The exact delimitation of the lesion to the posterior column was very obvious during the cutting of the preparations in the microtome. The condition of the wound in each case was closely similar, so that one description will serve for all. It consisted simply of a microscopic slit extending from the surface of the cord to the posterior commissure, and containing a little wound-serum and debris of damaged nerve-fibre, the mouth of the wound being always closed by young but firm connective tissue. The appearance of the sides of the wound-cavity, although so extremely small, showed clearly that there was some degree of tension in it; tension therefore was present in the interior of the cord, a factor that seems to me of some pathological importance in the correct estimation of the effects of such wounds of the cord. The chief point, however, which calls for particular notice in this method of research is the degeneration in some of the parts of the cord contiguous to the seat of operation, and of which a great deal has already been said. We may conveniently discuss this important point under the headings *Nature*, *Locality*, and *Causation*. The part which it plays in producing Prof. Schiff's result I have already referred to at full length, therefore now only wish to draw attention to the details of its existence.

(1.) *Nature.*—Prof. Schiff, it will be remembered, insisted on an interval of a few days being allowed to elapse,

in order, as he said, that the degeneration might reach the cortex. Now how far the ascending degeneration of the posterior columns gets in four or five days, I do not know, not having traced the course of the degenerated fibres; but this much is perfectly evident, that it does not arrive at, and cause the destruction of, the ganglion-corpuscles in the sigmoid gyrus, as Prof. Schiff would have us believe, for, microscopically, these corpuscles show no degenerative changes. But it is plain that this interval allows of the degeneration in the motor tracts of the spinal cord about to be described. Now the nature of this degeneration is very simple, consisting, as it does, merely in the swelling of axis-cylinders and myelin sheaths, so as to form the well-known and classical picture of the histological changes produced in nerve-fibres by acute "inflammatory" mischief within that space of time. In the accompanying drawings, where the shading has been marked darkly, there it is to be understood that the degeneration was very complete, that is to say, that all the nerve-fibres in the degenerated area were destroyed. To prevent misapprehension on this point, it will be well to draw attention to the fact that this complete destruction was only to be found in the direct cerebellar tract, the crossed lateral tract, and the direct pyramidal tract of the same side as the wound in the cord. I mention this particularly, since in Nos. 2 and 5 it will be noticed that the degeneration is necessarily represented diagrammatically as being spread over the whole lateral column, because degenerated fibres could be detected throughout that half of the section, although complete destruction was limited to the tracts above noted. It is scarcely necessary to describe the histological details at length, since, as has been stated above, they presented the ordinary appearances. It will make, however, this account more complete if I briefly state the chief facts:—

Changes in—

(A.) *Nerve Fibres.—Axis Cylinders.* The axis cylinders of the fibres involved in the degenerative process remain unaltered at first, since they can be detected in the centre of the swollen myelin (*vide* infra).

Soon, however, they atrophy, and in the majority of instances the space (dilated) in the neuroglia formerly occupied by the fibre is simply filled with a deeply staining myelin globule. The continuity of the fibre is thus completely interrupted.—*Myelin.* The myelin exhibits the first effects of the damaging process. It swells, and at first is but feebly stained by aniline blue-black; then it swells more, so as to fill the space in the neuroglia, and it stains nearly black. Finally, it appears to shrink again, and is absorbed or, as a rule, simply drops out in the preparation of the section.

(B.) *Neuroglia.*—The changes in the neuroglia, as might be expected, are not very marked, owing to the shortness of the period of time during which they might develop. In fact, as regards the fine supporting trabeculæ of connective tissue running with the capillaries, the only notable point about them is that they take the staining more deeply on the degenerated areas. The fine meshwork between them appears unaltered, save by the distension of the fibre spaces before referred to. As is noted in the description of each of the spinal cords at the seat of operation, the grey cornua in the neighbourhood of the wound are in most cases infiltrated to a greater or less extent with leucocytes, and there is also under these circumstances a distinct appearance of œdema.

(C) *Blood-Vessels.*—The degenerated area in every case was very anæmic, and in one instance, No. 4, this was extremely marked, while in the right half of the cord the vessels were full of blood. The importance of this fact will be discussed directly. (*vide Causation*, infra.)

(2.) *Locality.*—The exact locality of the degeneration is shown in the accompanying sketches of the cords, *vide* pp. 56, 57, but attention may be drawn with advantage to one or two cardinal facts determining its distribution.

Thus at the first glance it will be seen that the mischief *is almost confined to the side of the cord operated upon.* Next, that it is most marked in the closely adjoining posterior third

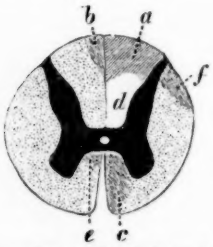

Fig. 1.—*Central Canal* distended, and contained exudation and blood. *The left posterior cornu* was infiltrated with leucocytes for half its breadth, and in some sections through its whole thickness.

Septic Case.—Pia mater was thickly infiltrated with leucocytes (pus). Schiff's result, marked.

a = Left posterior column destroyed, *i.e.* track of knife. *b* = Small portion of right p. c. degenerated. *c* = Whole direct pyramidal tract of left anterior column very greatly degenerated. *d* = Cavity of wound. *e* = Slight degeneration of direct pyramidal tract of right anterior column. *f* = Degeneration of ascending direct cerebellar tract.

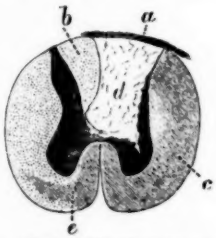

Fig. 2.—*Central Canal* was normal. It is not represented in the figure, which is a copy of the microscopical preparation. *The left cornua* were much infiltrated with leucocytes, and here and there patches of extravasated blood.

Aseptic Case.—Schiff's result, marked.

a = Scar tissue closing wound. *b* = Right posterior column (⅓rds.). *c* = Area of degeneration on left side involving whole antero-lateral column. *d* = Wound cavity full of debris. (*Note.* The damage done appears much larger, as already explained, owing to the friability of the "inflamed" grey substance.) *e* = Degeneration in the direct pyramidal tract of the right anterior column.

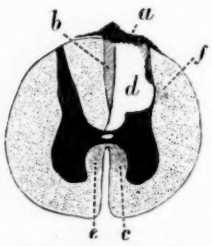

Fig. 3.—*Central Canal* contains blood clot.

Aseptic Case.—Schiff's result, very slight.

a = Scar tissue plugging wound. *b* = Remains of left posterior column destroyed. *c* = Degeneration, very slight in left anterior column. *d* = Wound cavity. *e* = Degeneration of right anterior column, slight. *f* = Slight œdema (?) of crossed lateral tract.

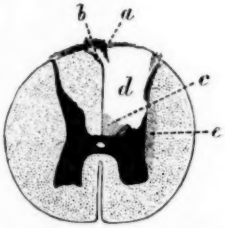

Fig. 4.—*Central Canal* moderately distended with exudation. *The left posterior cornu* was infiltrated with leucocytes at the notch represented in the drawing.

Aseptic Case. Schiff's result, extremely slight.

a = Scar tissue closing wound. *b* = Small angle of right posterior column cut away. *c* = Small patch of degenerated left posterior column. *d* = Cavity of wound. *e* = Small vertical patch of degeneration just outside cornu of grey matter.

of the lateral column. Thirdly, that the next place of greatest frequency is the direct pyramidal tract in the vertical line of the wound. The explanation of the greater liability of these areas to be affected will be discussed directly, *vide Causation*,

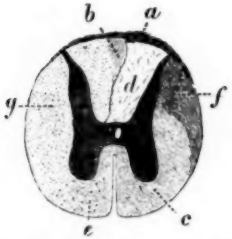

Fig. 5.—*Central Canal* normal. Whole cord, but especially left grey cornu, is infiltrated slightly with leucocytes. Septic Case. — Cord œdematous. Schiff's result, excessively marked, see p. 6.

a = Scar tissue and pia mater. b = Damaged portion of right posterior column. c = Degenerated left antero-lateral column. d = Cavity of wound filled with debris of left poster or column. e = Degenerated direct pyramidal tract in right anterior column. f = Degeneration of crossed lateral tract in left half of cord. g = Very slight degeneration in right crossed lateral tract.

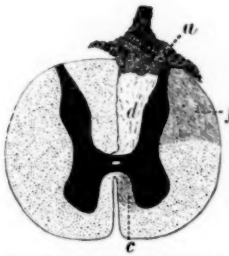

Fig. 6.—*Central Canal* normal. Left cornu infiltrated with leucocytes. Aseptic Case. Schiff's result, marked.

a = Scar tissue. d = Cavity of wound filled with debris. c = Degeneration in direct pyramidal tract of left anterior column. f = Crossed lateral tract and cerebellar tract much degenerated to exact limit shown.

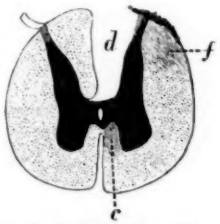

Fig. 7.—*Central Canal* normal. Aseptic Case. Schiff's result, very slight.

d = Wound cavity. c = Degeneration in left anterior column. (This was extremely limited.) f = Well-marked degeneration of the cerebellar tract, extending but slighily into left crossed lateral tract.

infra; but I must now state that the degeneration is only marked exactly opposite the wound in the cord, and only extends a millimetre above and perhaps two millimetres below that point. This narrow limitation of the degeneration

proves it to be an entirely local effect of the operation. I mention this, because it might possibly be imagined by some that ascending degeneration might, as Schiff believes, go up to the sigmoid gyrus, destroy the corpuscles there, and then come down the pyramidal tract. In each case I have examined the cord microscopically, not only just above and below the seat of operation, but also opposite the 1st and 2nd cervical nerves, and in no instance was any degeneration observable in the motor tracts at the summit of the cord. We may now discuss the causation of this degeneration.

(3.) *Causation.*—In the first place it is to be remembered that, with the exception of Nos. I. and V., each experiment was absolutely aseptic, and therefore we may set aside sepsis as a cause of the degeneration. From a consideration of the facts that the degeneration was most marked in proportion to

1. The amount of distension of the wound-cavity, in the cord and this means also the density and size of the cicatricial plug closing it;

2. The amount of anæmia and damage to blood-vessels; it is clear that we have suggested to us two ways in which this overlooked source of fallacy may be caused. These two possible ways are, tension in the cord, and interference with the blood-supply. In the first place, tension will obviously tell most heavily on the neighbouring crossed lateral tract. In the second place, the whole vascular supply to the left posterior quadrant of the cord will be interfered with by the operation, and further than that, a certain amount of thrombosis may occur which would reach the median artery, and so cut off the supply to the direct pyramidal tract. It would not be profitable to go over again the evidence offered in the foregoing pages, proving that both of these conditions follow the division of the posterior column, but it is worth while to note, that, as expressed also in the Table on p. 62, where these conditions were only slightly present the results as described by Schiff were very feebly marked, and vice versâ.

Having now seen clearly that a little localised patch of

acute degeneration (plus tension in the cord) attacks invariably, though in very different degree, the motor tracts in the spinal cord on the same side as a wound of the posterior column, and that such a patch and such pressure necessarily form a block, offering greater or less resistance to the passage of motor impulse from the excitable cortex, we may turn to review the results obtained by stimulating the cortex in these experiments, as directed by Prof. Schiff. It will be remembered that in every case the *left* posterior column was the one divided. In the first two experiments I first exposed and tested the right excitable area, and then subsequently (*i.e.* ten or fifteen minutes later) the left. In the remaining five experiments I first removed the bone from the dura mater over both the motor cortices, and then raised the dura mater so that the exposure of the two sides was practically simultaneous, while in testing, the electrodes were first tried on one side and then on the other. This slight difference in the mode of treating the cortex on the two sides, as might be expected, made no difference in the results obtained. As will be seen from the subjoined Table, the amount of movement of the fore-limb of the left side as compared with that of the right side, when the right and left sigmoid gyri were respectively stimulated, varied inversely as the amount of degeneration visible in the motor tract of the cord opposite the seat of operation. Consequently it is erroneous to describe the diminution of movement as being due to a decrease in the excitability of the sigmoid gyrus, when it is really owing to the fact, that of course the swollen axis-cylinders in the degenerated lateral columns are incapable of transmitting the energy discharged from the irritated cortex. This experimental result is an interesting illustration of the clinical observation of paralysis from acute "inflammatory" conditions in the spinal cord. It will not be necessary to take up space with the minute details of all the cases experimented upon, but I may give short notes of Experiment V. as illustrating, first, the most extreme condition of Prof. Schiff's result; secondly, the fact that (as I have mentioned before) this result was obtained to such a very marked degree because, owing to the wound becoming septic, the degeneration was more

extensive; and finally, the general method followed. The following is an abstract of the details of this experiment.

EXPERIMENT V.—DOG, ADULT MONGREL TERRIER.

Sept. 11*th*, 1885.—Left posterior column divided (according to method described above). Animal asphyxiated by ether, but recovered by artificial respiration.

12*th*.—Dog apparently well.

13*th*.—Marked paralysis of left fore-paw. Lies habitually on left side.

In attempting to walk, frequently falls to left side (hemiplegic).

14*th*.—During previous night, symptoms of spinal meningitis noted.

At 2.20 P.M. both fore-legs rigid, general rigidity of trunk, &c., with the other classical symptoms of meningitis. Dog immediately anæsthetised with ether.

2.45 P.M.—Both sigmoid gyri exposed within two minutes, and then stimulated as above described.

(I may here take this opportunity of stating, that in this series of experiments the apparatus of stimulation were an ordinary Du Bois Reymond induction coil, one Daniell cell [in one case, two cells were used] and usual platinum electrodes. The divisions indicating the distance of the secondary from the primary coil are given in centimetres; the strongest current, therefore, being at zero.)

Current.	Right Sig. Gyrus.	Left Sig. Gyrus.
10 cm.	Nil.	Nil.
8 cm.	Nil.	Nil.
6 cm.	Flickerings in left trapezius.	Fibrillar twitchings in right trapezius and protrusion of limb with extension of fore-paw.
5½ cm.	Ditto.	Ditto, stronger.
Interval, *i.e.* rest of 5 minutes.		
5½ cm.	Ditto, no spread.	Ditto, and spread to head, and right lower limb.

"The difference was so extremely marked as not to need further investigation."

In fact a superficial examination would have led one to

state that the right sigmoid gyrus, as far as the centres for the limb- and trunk-muscles respectively were concerned, was inexcitable. As a matter of fact however, in this, as in all other experiments, the right cortex was perfectly excitable, as evidenced by the active movements of the facial and cervical muscles as low as the distribution of the fourth cervical nerve. I have already described the condition of the spinal cord in this case (see page 57), in which description it will be remembered that the degeneration of the motor tract was most marked. This case will thus serve as a typical illustration of the mode in which I believe Prof. Schiff's result is always produced. Of course it was impossible in this instance that the nerve energy should reach the left limb muscles.

The rest of the facts concerning the production of Schiff's result, viz. the diminution in the usual amount of muscular movement evoked by stimulation of the opposite sigmoid gyrus, so invariably illustrate (*vide* Table, p. 62) the fact alluded to before, viz. the variation of degree of loss of power, being proportionate to the damage of the lateral columns, that but little addition is necessary to the details just enumerated. There are, however, one or two points which are of considerable interest. In the first place, in those cases where Schiff's result was scarcely obtained at all it was only obvious when powerful currents were employed. Moderate, but very adequate stimuli called forth the usual movements on both sides, if a little different in force, but with such a stimulus as a current at 5 cm. this difference in violence of the movement became very apparent. The second point is this, that in three instances there was noted very definite clonic tremor (post-ethereal) of a rate of ten to twelve per second, which occurred in the left fore-limb first before invading the others. This fact seems to me worthy of note, since it appears to indicate that ordinary clonic tremor, so familiar clinically, is, as I suggested in my Brown Lectures, 1885, not the simple question of a rhythmical alternation of contraction of opposing groups of muscles, but that it (ankle-clonus, for example) is really the rhythmical contraction of a given muscle (*e.g.* the gastrocnemius) in response to a rhythmical discharge of the spinal motor corpuscles governing

that muscle, such corpuscles having been thrown into discharging activity by the sudden violent afferent impulse which is generated (according to the usual clinical method) by forcibly stretching that muscle, *e.g.* by flexion of the ankle. The pathological irritability of the motor corpuscles in my experiments obviously is set up by the changes I have described in the descending pyramidal tracts, an experimental condition comparable to the pathological one of sclerosis.

Conclusion.—The inference to be drawn from the above experimental examination of the question whether Schiff's method shows the posterior columns to ascend directly to the motor or excitable cortex must, I think, be that such direct connection is *not proven.* The investigation (the expenses of which were defrayed by a grant of 5*l.* from the British Medical Association) has elicited the fact, not, so far as I know, published before, that tension and anæmia in the cord will bring about marked local degeneration in the motor tract especially.

TABLE showing the relation between the DEGENERATION in the ANTEROLATERAL COLUMNS and the EFFECTS which were supposed to be due to LESION of the POSTERIOR COLUMN, this being in every case completely divided.

No. of Experiment.	Paresis.	Tactile Anæsthesia.	Schiff's result of stimulating opposite excitable area.	Degeneration in Motor tract.	Remarks.
I.	Present.	Could not be tested.	Marked.	Marked.	Septic.
II.	Present.	Present.	Marked	Marked.	Aseptic.
III.	Absent.	Absent.	Very slight.	Very slight.	..
IV.	Absent.	Absent.	Very slight.	Extremely slight.	..
V.	Almost Hemiplegic.	Could not be tested.	Very marked.	Extreme.	Septic.
VI.	Present.	Present.	Marked.	Marked.	Aseptic.
VII.	Absent.	Absent.	Very slight.	Slight.	..

NOTE.—When Schiff's result of want of movement following stimulation of the right sigmoid gyrus as compared with that following excitation of the left is set down in varying degree in the 4th column, it is to be remembered that the terms in which it is described are exact, since they are founded on the experimental data which are the measurements of the strength of the current in centimetres of the distance of the secondary from the primary coil; see p. 60.

ON PROFESSOR HAMILTON'S THEORY CONCERNING THE CORPUS CALLOSUM.

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In the last number but one of 'BRAIN' (October, 1885), a short preliminary note was given by me in reference to the view propounded by Prof. Hamilton, that the corpus callosum is not an interhemispheral commissure at all, but is the decussation of the cortical fibres in their progress downwards to become connected with the basal ganglia.

It is proposed in this paper to test the theory by the evidence afforded by the microscopical examination of very thin sections stained by Weigert's hæmatoxylin method, and by comparing the corpus callosum in man with that of the other mammalia, and especially with regard to the relative size between the anterior commissure and the corpus callosum in the lower animals.

I will at once begin with the evidence which microscopical examination gives us on this question.

With regard to the evidence of microscopical examination, it should be stated that my sections have been made chiefly from the monkey; for although many sections have been made from the brain of man, I have relied especially on those of the former, for the reason that the brains of these animals can be had absolutely fresh, and can be put into the hardening fluid directly after the animal is killed. Besides the serious difficulty of obtaining the brain of man fresh, it is too large to trace with accuracy the fibres to any distance, when viewed under the higher powers of the microscope. It would be well to say at once that I have not in any way relied on the naked-eye appearances, or the mere use of a magnifying glass, but all the sections have been examined under various powers

of the microscope. The brain of the marmoset has also been examined, and has proved very easy of manipulation, owing to its small size.

The brains taken from the monkey were treated as follows: the animal was killed with chloroform, and at once the brain was removed and put whole into a mixture of methylated alcohol and water (half and half) for 24 hours, then it was cut into pieces of ¼ inch thick—frontally, horizontally or sagitally, as the case might be—and put into bichromate of potash solution 3 per cent., for three to four weeks, and afterwards the hardening was completed by methylated alcohol. The pieces were imbedded in collodion and sections cut; they were then stained by Weigert's hæmatoxylin method, and passed through methylated alcohol, absolute alcohol, oil of cloves, and mounted in Canada balsam.[1] The marmoset's brain was stained in toto by Weigert's hæmatoxylin, and cut in paraffin, as described in 'BRAIN,' July, 1885.

In some cases the brains were sliced and put at once into bichromate-of-potash solution. On looking at a section cut from a brain prepared in the above way, the individual medullated fibres will be seen stained quite black, and their course can thus be well made out.

If we examine a frontal section taken from about the posterior part of the corpus callosum (Fig. 1) on a level with the middle of the pons Varolii, it will be seen that the corpus callosum (c c) on reaching the hemisphere on either side spreads out like a fan, sending fibres upwards to the convolutions at the vertex (c′ c′) and outwards to all the convolutions down to a certain level. Now this level is found in the monkey to be the upper part of the Island of Reil; I have examined most carefully with the high power of F Zeiss, but I have failed to find any transverse or oblique fibres of the corpus callosum below this point, they seem to stop suddenly. This fact is therefore at variance with Prof. Hamilton's view, who considers that many of the fibres of the crossed callosal tract end in the external capsule.

With regard to the internal capsule, I cannot find a single fibre which passes from the corpus callosum into the internal

[1] 'Fortschritte der Medicin,' 1885, No. 8.

capsule; the lowest fibres of the corpus callosum run horizontally outwards to the upper part of the Island of Reil, at right angles to the fibres issuing from the external and internal capsules; the corpus callosum does not pass into either of the capsules, in the monkey or in man.

The fibres issuing from the internal capsule (C I) course upwards, at first at right angles to the callosal fibres, but higher up they cross them obliquely (C′I′), and can be traced the whole way from the capsule to the grey matter of the convolutions of the same side. The most internal fibres from the internal capsule have a more curved course than the more external ones; for three-fourths of their course across the callosal fibres they are easy to trace, but after this point it is more difficult to follow them; but by comparing different sections and using high powers they appear to go to the lower part of the marginal convolution; in my opinion they certainly do *not* go into the corpus callosum; these fibres are the only ones which present any difficulty in ascertaining their destination, as all the other fibres from the internal capsule can be easily traced to the convolutions of the same side.

From the external capsule (C E) only a few fibres come from the part internal to the claustrum and ascend with the fibres from the internal capsule on their way to the convolutions of the same side.

These fibres from the external capsule are situated externally to the main mass coming from the internal, but they are crossed by fibres (O P) passing from the internal capsule to the convolutions on the upper border of the Sylvian fissure. The fibres from the external capsule do not, in my opinion, come into any relation with the corpus callosum except to cross at right angles to its lower fibres, and to run parallel with its upper fibres on their way to the vertex; the two are certainly not continuous.

The brain of the marmoset presents very important evidence in favour of the commissural theory of the corpus callosum. I have managed to stain this brain in toto by Weigert's hæmatoxylin,[1] and have cut the whole brain frontally into sections, from the optic chiasma in front to the posterior part

[1] 'Brain,' July, 1885.

of the pons behind; every section has been looked at, and only those have been mounted in series which present any naked-eye difference to those preceding them; by this means we get a series of over two dozen sections showing all gradations of change, between the optic chiasma and the hinder part of the pons. On examining all these sections with the highest powers of the microscope, I have failed to find a single case where the fibres of the corpus callosum pass into the internal capsule. As has been mentioned in the preliminary note,[1] the brain of the marmoset differs from that of the other apes in the fact, that the gyrus fornicatus does not exist as a separate convolution, lying on—or appearing to spring up from—the upper surface of the corpus callosum, but it is more blended with the convolutions at the margin of the longitudinal fissure, so that in a frontal section the medullated antero-posterior fibres of the gyrus fornicatus (cingulum) form a projection at the lower part of the inner face of the centrum ovale above, and to the inner side of the peripheral end of the corpus callosum. This simplifies matters very much, and we do not have any fibres from the internal capsule making their way up through the corpus callosum before it begins to spread out.

This disposition of the gyrus fornicatus in the marmoset, and the fact, alluded to further on—that the fibres of the internal capsule have a course which is directed almost quite straight upwards and inwards—have, I think, a most important bearing in understanding the relations of the corpus callosum and the internal capsule. I believe that the large curve which the fibres of the internal capsule in man make, with the concavity of the bend towards the corpus callosum, has led Prof. Hamilton to suppose that the internal capsule actually passes into the corpus callosum. He has shown these curved fibres in his figure in the 'Journal of Anatomy and Physiology,' but he has not drawn them actually passing into the corpus callosum. In the monkey these fibres from the internal capsule are less curved; while when we come to the marmoset we find them almost straight, and at right angles to the horizontal axis of the corpus callosum.

[1] 'Brain,' October, 1885.

In the marmoset the arrangement in all these sections is this: the corpus callosum spreads out its fibres like a fan before reaching the centrum ovale, and sends the upper part of this fan upwards, outwards, and then inwards to just above the gyrus fornicatus; the middle part through the internal capsular fibres to the cortex, while the lower part runs downwards and outwards to the cortex, meeting and crossing the fibres coming up from the internal capsule, and here a well-marked decussation occurs between these different sets of fibres.

The fibres from the internal capsule have nearly a straight course upwards and inwards to reach the convolutions at the vertex of the same side; they do not present the curve, concave inwards, which is seen in the other apes and man, and in no place do they pass into the corpus callosum, nor does this latter send any fibres into the internal capsule.

Prof. Hamilton has stated that the pyramidal tract, alone of the fibres forming the corona radiata, does not pass into the corpus callosum to decussate to the opposite side, but that the rest of the fibres, anterior and posterior to the pyramidal tract do pass into the corpus callosum. Now if this were the case we should find at the borders of the pyramidal tract a sudden change in the disposition of the fibres coming up from the external and internal capsules, they would form a sharp line of demarcation on each side of the pyramidal tract which would show where the crossed callosal tract begins. With regard to the corpus callosum itself, at the level of the pyramidal tracts (in frontal sections), what is its function there? It cannot be a decussation, as we are distinctly told that the pyramidal tracts are alone exempt from the callosal decussation,[1] and it is not a commissure between the hemispheres, it surely ought to be absent altogether at this point, and we should have a blank space opposite the pyramidal tracts. I must maintain, from an examination of my marmoset sections which were made in front of the optic commissure, as well as from those between this point and the hinder part of the pons, that there is absolutely no difference in the relation of the corpus callosum to the capsular fibres; there is no change of structure at the anterior or posterior margins of the pyramidal tracts

[1] 'Liverpool-Medico-Chirurgical Journal,' Jan. 1886.

different to what is seen in a section made through the centre of these tracts.

To show the arrangement of the corpus callosum and the internal capsule in the anterior part of the brain, I have cut horizontal sections of the brains of man, the monkey and marmoset, prepared in the way mentioned above, and stained with Weigert's hæmatoxylin. The relation of the parts is seen in Fig. 2, which is a horizontal section through the left genu of the corpus callosum and the anterior fibres of the internal capsule. The under part of the genu of the corpus callosum is here seen to be divided into two parts, an anterior and posterior; the anterior part (c′ c′) begins in the extreme tip of the frontal convolutions, and runs backwards and then inwards to cross over to the corresponding part on the opposite side, forming a true commissure between the two frontal convolutions; the posterior part (c c), quite distinct and separated off from the anterior, passes from the middle line of the corpus callosum, and runs outwards till it meets with the fibres coming from the internal capsule (c i). This latter has its fibres in parallel bundles until it has passed the lenticular and caudate nuclei (n l n c), it then reaches the medullary white matter, and sends its fibres anteriorly, externally, and posteriorly. The anterior fibres (c′ i′) go to the tip of the frontal convolutions and form a very sharply defined bundle, which can be traced the whole way into the frontal convolutions of the same side; the whole of these anterior fibres can be seen passing through the fibres from the posterior part of the germ of the corpus callosum (c c), with which they make a sharp angle and a well-marked decussation (d); while the posterior fibres of the corpus callosum make their way to the cortex in front of the fissure of Sylvius.

It is quite evident that the anterior fibres go straight from the internal capsule to the tip of the frontal convolutions, and in no case can I find the fibres of the corpus callosum passing into the internal capsule; the two parts of the corpus callosum are quite distinct, and no decussation in the middle line takes place between them.

The decussation of the corpus callosum with the internal capsule, which is so evident at the angle where these fibres of

the internal capsule and corpus callosum first meet, can also be seen by a high power to take place in the mass of white medullary substance in front of, and opposite to the point of entrance of the internal capsule.

From comparative anatomy there are several important facts which throw much light on the structure of the corpus callosum, and this is especially the case when comparison between the relative size of the corpus callosum and the anterior commissure is made in different animals.

In man the diminutive size of the anterior commissure as compared with the corpus callosum is well known; but already in the monkey it can be seen in a frontal section that the vertical cross area of the anterior commissure is almost as large as that of the corpus callosum, although of course the longitudinal area of the latter is many times in excess of the former. This relation between the two commissures was pointed out by Prof. Flower in a most interesting and valuable paper,[1] where he calls attention to the excessive development of the anterior commissure, and the small size of the corpus callosum in the marsupia. Prof. Flower's paper is illustrated with drawings of the frontal and sagittal position of brains of various animals, from man down to the marsupia and monotremata.

I have lately, through the kindness of Mr. Beddard, prosector at the Zoological Gardens, obtained a brain of the large kangaroo (*Macropus major*); this has been hardened and stained by the above methods, and frontal sections have been cut through the corpus callosum and the anterior commissure, so as to show both at once. In this animal, as described by Prof. Flower, the corpus callosum is very small indeed, especially when seen frontally, and is a mere thread—so small that Prof. Owen hardly considered it to be a corpus callosum; but on this point I venture to think that there can be no doubt that Prof. Flower's opinion is correct. When this brain is stained by Weigert's method, the corpus callosum is seen to pass from one gyrus fornicatus to the other, and to act as a commissure between these convolutions only. On the other hand, the anterior commissure is enormously developed, and occupies one-fourth of the whole vertical height of the brain;

[1] 'Philosoph. Transact.,' 1865, Pl. xxxvii. and xxxviii.

its fibres form a thick band in the middle line, and on each side it divides into two bands which run into the hemispheres on either side, and acts as a commissure between the upper convolutions, as well as between the lower of either side. The bands of fibres can be traced completely across from side to side without decussating.

The difference of the size between these two commissures in different animals seems evidently due to the difference in the size of the parts of the brain they have to join; in the kangaroo, the temporo-sphenoidal convolutions are enormously developed, while the frontal region is very small, and hence the small size of the corpus callosum as compared to the anterior commissure. This condition in the kangaroo has a very important bearing on the structure and function of the corpus callosum in man and the higher mammalia. In the kangaroo, it is perfectly impossible that the thin shred of a corpus callosum can be the path of decussion of the crossed callosal tract, coming from the internal capsule; besides which, the fibres of the corpus callosum can be seen to end in the gyri fornicati. It is evident that the only path left for the crossed callosal tract must be the anterior commissure; but this is in front of the pyramidal tract, and the anterior fibres of the corona radiata are here seen to be cut transversely, and to have no connection with the anterior commissure.

This point alone seems to me to be very strongly against Prof. Hamilton's theory of the corpus callosum.

He has asked the question, How do animals which have no corpus callosum manage to perform combined movements of the two sides, and he considers that the absence of this body militates against the theory that it is a commissure between the two hemispheres. I think this question is sufficiently answered by the fact, that as we descend the mammalian scale, the anterior commissure is developed in proportion as the corpus callosum diminishes, until in the marsupialia the former attains the enormous size seen in the kangaroo.

In the kangaroo neither the corpus callosum nor the anterior commissure can be considered as a crossed callosal tract, and it cannot, I think, be conceived that in the lower mammalia the brain should be formed on a type totally different to that

of the higher animals; this must be contrary to all ideas of evolution.

In the 'Liverpool Medico-Chirurgical Journal' for last January, Prof. Hamilton has done me the honour of referring to my preliminary note in 'BRAIN,' October, 1885. He there complains that I have not followed a single one of the methods he employs, and that the clarifying process I use is so coarse as in itself to destroy any chance of getting a view of the continuity of the crossed callosal tract, and that I shrink all the beauty out of the callosal fibres by employing spirit as a hardening reagent.

The method which I use—Weigert's hæmatoxylin—has the property of staining all the medullated fibres of the brain, and is so delicate and perfect as to show completely the finest fibres of the cortex, and present such a delicate network there, that they can only be fully appreciated by using high powers, as the F Zeiss. And we are to suppose that these minute fibres are to be everywhere stained in my preparations, and yet the crossed callosal fibres of the bulky corpus callosum—which Prof. Hamilton can see with an ordinary magnifying glass—are in some mysterious manner shrunk away by using methylated alcohol, after hardening in bichromate of potash for a month, and by my coarse clarifying agent,—oil of cloves! With regard to this latter reagent, I can positively state that no disappearance of fibres occurs, as I examine the sections under a low power first in water, then in alcohol, and finally in the oil of cloves; there is only this difference, viz. that in the oil of cloves the details are more clearly brought out, owing to its well-known properties of clearing the specimen. Further comment is, I think, unnecessary.

I have not employed Prof. Hamilton's method; as I consider that swelling and separating the fibres by the action of potash and gelatine must tend to disturb their normal arrangement much more than alcohol or oil of cloves, and I should much like to know whether by his method he is able to show the finest plexus of fibres in the cortex, or, if they are visible, whether they are seen as clearly as they are by Weigert's hæmatoxylin method; until that is possible I shall continue to employ the latter method.

Although I am charged with "shrinking all the beauty out of the callosal fibres by employing spirit as a hardening reaction," what do we find in the description of Prof. Hamilton's process in 'BRAIN,' July, 1883, p. 214? We are actually told there to put the pieces of brain into pure methylated spirit, to finish the hardening after Müller's fluid,—precisely what I have done with my preparations. The use of Weigert's säure fuchsin has been alluded to in some of Prof. Hamilton's papers, but as he soaks his preparations for four days in water, and then in an aqueous solution of mucilage and syrup for two weeks or more, it is quite impossible that he has obtained the proper reaction. I make this statement with due caution, as, having had the advantage of working with Prof. Weigert at the time he made this discovery, and being, I believe, the first to put it to practical use in my work on the cerebellar cortex at the Physiological Laboratory at Leipzig, where I used this dye daily for six months, I can endorse the canon that Prof. Weigert has laid down, viz. that on no account may the pieces of tissue be put into water before using the säure fuchsin.

Prof. Hamilton states that I have not employed oblique sections as he directed, and yet the only two drawings which I have seen published—in the 'Journal of Anatomy and Physiology'—are perpendicular transverse sections; I have however made obliquely frontal sections of the monkey's brain, but have failed to see the crossed callosal tracts.

The two drawings, mentioned above, are drawn presumably the natural size of the human brain, and although the crossed callosal fibres are figured, they are not drawn (in Plate xxii.) as actually passing into the corpus callosum; they end abruptly, and might equally well pass on to the cortex of the same side.

Will Prof. Hamilton publish a *microscopical* drawing of these fibres? As in his papers, with the exception of that "on the Corpus Callosum in the Embryo," little allusion is made to the microscopical appearances, and in his last paper in the 'Liverpool Medico-Chirurgical Journal,' he states that he "cannot at present enter into the technicalities of the microscopic examination."

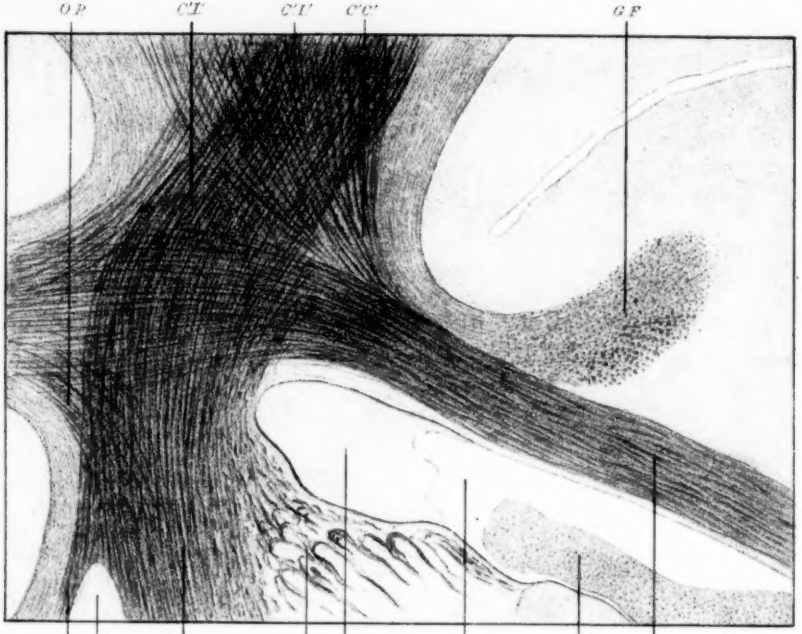

Fig 1

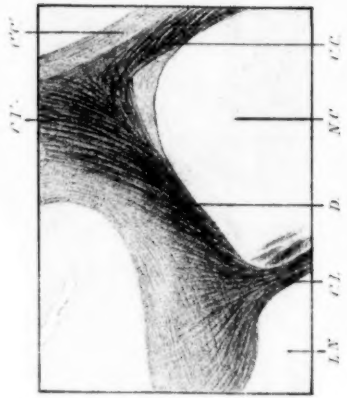

Fig 2.

Dentsson & Co. Lith.

From a careful examination of this question from the point of view of anatomy and comparative anatomy, I can only come to the conclusion, that the generally received idea of the corpus callosum is quite correct.

The corpus callosum I consider to be a commissure between the two hemispheres; and the fibres of the internal and external capsules, in my opinion, do not pass into the corpus callosum to decussate to the opposite cortex; no distinct decussation can be demonstrated in the middle line of the corpus callosum, and the corona radiata does exist as such, and is not a nonentity, as Prof. Hamilton would wish us to consider.

EXPLANATION OF THE PLATE.

FIG. 1—is a *frontal* section through the left centrum ovale of a monkey's brain, and shows the decussation between the corpus callosum, and the internal and external capsules. The section is made at the vertical level of the middle of the pons Varolii, and through the hinder part of the corpus callosum, just in front of the splenium. The preparation was stained with Weigert's hæmatoxylin; and the drawing is in no way diagrammatic.

Magnified 10 diameters, and reduced.—C.C. left half of corpus callosum.

C'.C'. fibres of corpus callosum to convolutions at vertex.—C.I. Internal capsule.

C'.I'. fibres of internal capsule to convolutions at vertex, decussating with corpus callosum.

C.E. external capsule sending fibres to vertex, decussating with corpus callosum.

O.P. fibres from external and internal capsules to supra-marginal and ascending parietal convolutions.

L.N. lenticular nucleus.—C.N. caudate nucleus.—O.T. optic thalamus.

L.V. lateral ventricle of left side.—F. left half of fornix.—G.F. gyrus fornicatus.

FIG. 2—is a *horizontal* section through a portion of the anterior left half of a monkey's brain, and shows the decussation between the corpus callosum and the internal capsule. Prepared like Fig. 1.

Magnified 10 diameters, and reduced.

C.C. the left half of the genu of the corpus callosum, the hinder part of which turns backwards to decussate with the internal capsule.

C'.C'. the anterior part of the genu of the corpus callosum, which joins the anterior frontal lobes.

C.I. anterior fibres of the internal capsule.

C'.I'. fibres of internal capsule to anterior frontal lobes, after decussating with corpus callosum.

D. point of decussation of internal capsule and corpus callosum.

N.C. left caudate nucleus.—L.N. left lenticular nucleus.

Clinical Cases.

CASE OF INJURY TO SKULL.—TREPHINING FOR EPILEPTIFORM ATTACKS.—REMOVAL OF DEAD BONE.—DEATH.

BY RICHARD DAVY, M.B., F.R.S.E.,

Surgeon to the Westminster Hospital.

With a NOTE *on the* PHYSIOLOGICAL *and* MEDICAL ASPECTS *of the* CASE,

BY A. HUGHES BENNETT, M.D., F.R.C.P.

T. N., æt. 22, gasworker, single, was admitted into the Westminster Hospital on December 22, 1884, with a small scalp-wound, and injury to calvarium over the left parietal bone. Mr. Waller, the house surgeon, found that his probe entered the skull vertically for an inch downwards, and free bleeding was present. The patient stated that his head had been knocked by a falling iron chain; and that a hook at the end of the chain had swung, and caught him on the side of his head. His gait was unsteady and uncertain; he was quite conscious, and was at once placed quietly in bed. No signs of compression; a feeling of numbness of right side. He went on well, without any marked change, until Jan. 5, 1885. He then had marked twitchings of both arms and legs, followed by two epileptiform attacks; and on recovering, partial paralysis of his right face, right forearm, and right leg supervened; accompanied with a feeling of numbness of the right leg and arm. No paralysis of sphincters. My colleague, Mr. Thomas Bond, seriously discussed the propriety of trephining the skull over the wound; but postponed immediate operation.

Jan. 9th, 1885.—His urgent symptoms have subsided; so I restricted his diet to milk, one egg, bread and butter, and tea, and ordered open-air treatment to the wound, with cleanliness. I visited him daily during January, and with slight variations he continued to improve.

Feb. 2nd.—12.15 A.M. A marked epileptiform fit, followed by sickness. 3 A.M. Another, of 7 minutes' duration; cephalalgia, twitchings of both legs, especially the right. 7.30 A.M. Another fit, lasting 4 minutes. 11.22 A.M. Another fit, lasting 3 minutes.

At 1 P.M. I saw the patient in a fit, in consultation with my colleague Dr. Hughes Bennett; and we agreed to trephine over the wound at once. The man was chloroformed; a small exploratory trephine was used first at the edge of the original wound, and the circle of bone removed, and carefully examined on its dura-mater aspect, to see if the vitreous table had been broken by the accident. On finding this quite sound, a large trephine was used at the opposite part of the edge of the wound; on removal, this portion was found to be chipped irregularly, and marked the original injury; division of the dura mater permitted eight pieces of loose bone to be taken out of the brain substance; the deepest being two inches below the surface of the skull cap. My little finger carefully introduced through the trephine hole could feel the brain pulsating, but could detect no more detached fragments.

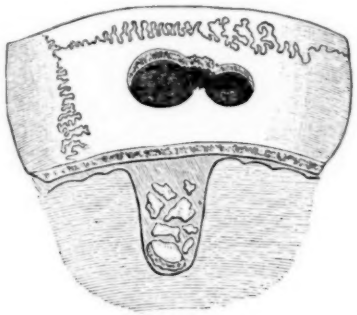

FIG. 1.

The diagram (Fig. 1) shows well the site of the original wound, trephine apertures, and the spiculæ. Some free hæmorrhage occurred, which was allowed to escape; for no dressings were used. The man slowly recovered from the chloroform, and had no fit until the next morning at 3 A.M. The patient was actually in a fit at the time of operating.

Feb. 3rd.—During the day had seven epileptiform attacks, each lasting a few minutes. These were preceded with marked twitching of the right arm; which the patient asked to be restrained. This day at 5 P.M. Dr. Hughes Bennett ordered him potass. bromid. gr. xxx. in water every six hours.

4th.—Good night, no fits; no marked headache. Pupils act equally. Right arm weak.

5th.—On account of diarrhœa the bromide of potassium was discontinued. Wound discharging; but its upper part has healed by first intention.

7th.—Pain in the right side of his chest and abdomen. Dry pleuritic rub. Muscular movement of his right arm has much improved.

8th.—Restless night; complains much of pain in his right arm and his chest. Posterior base of right lung is dull; dyspnœa; and friction-rub plainly to be heard. No headache.

10th.—Discharge from wound less; no fits. Restless from cough and pain at his right chest. States that he has had previous similar attacks.

12th.—He passed a good night; but his chest trouble is increased, crepitant râles heard all over his right chest; increase of vocal resonance, and frothy expectoration tinged

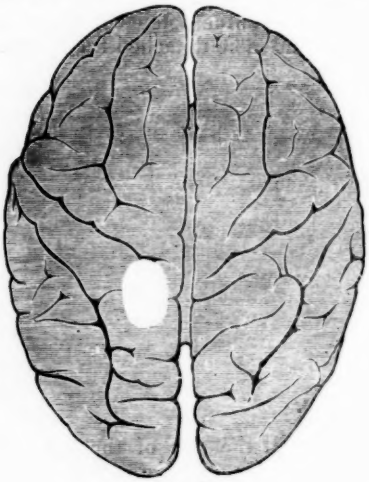

FIG. 2.

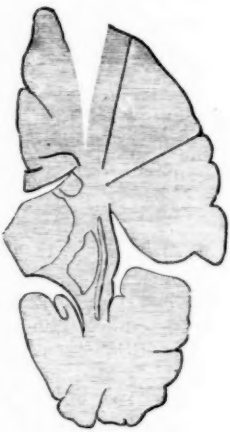

FIG. 3.

with blood. His face is pallid; his lips blue. Tongue dry, brown and raspy. Pulse 126. Respirations 48.

16th.—His symptoms became more marked; dyspnœa increased; and prostration intensified. He has not complained of any headache; his wound has given no trouble, and is entirely healed, except at one small point. He has not lost consciousness since February 3rd, 1885; and died, in a calm, rational, and exhausted condition at 12.10 A.M.

Post-mortem, February 18th.—An examination of the *head only* was allowed.

There was a scar on the left side of the vertex, which had recently healed, except at a point large enough to admit a small

probe. This scar was situate over the left parietal bone, 7½ inches from the bridge of the nose, and about 1 inch to the left of the vertex. There was only a little discharge, no smell; and no hernix cerebri. The calvarium was carefully sawn off and removed. The brain surface was adherent around the trephine apertures by organised lymph, and tough fibrous tissue. Imbedded in this, and intimately adherent to the dura mater, was a shell of vitreous table, as large as a canary seed. There was no surrounding cerebritis, nor meningitis. The superior longitudinal sinus was severed; and its nasal end was plugged with a firm fibrinous decolorised clot. A ragged wound, large enough to admit a small finger, descended into the brain substance; this was situated at the upper part of the ascending parietal convolution, and immediately in front of and partly involving the paracentral lobule. There was no pus present in the brain, nor any spiculæ of bone. The exact site of this brain lesion is shown in the two plates.

There were marked evidences of mustard-poultices and of dry cuppings over both sides of the chest, and intercostal bulging with dulness, on percussion, over the right half of the chest; but no further examination of the body was allowed.

Remarks by Mr. R. Davy.—By this injury certain results were produced of a localised character. For fourteen days after the accident the patient was free from fits, and then within three days he had three marked epileptiform attacks. After an interval of twenty-five days the attacks recurred, and operative interference was therefore considered necessary.

The treatment of the wound by the open method appears to be satisfactory, no hernia cerebri resulted, and healing was evidently all but secured. The chest affection appeared to be repetition of an old attack; but unfortunately the denial of a complete post-mortem leaves this man's variety of pleurisy and pneumonia a doubtful point.

He died fourteen days after the operation; and in my own experience, chest complications (after injury to the brain) appear to be a common sequel.

I must express my obligation to Dr. Hughes Bennett for his assistance rendered in this trying case, and I have also requested him to add his own postscript to the preceding notes.

Note, by Dr. Hughes Bennett.

Mr. Davy having described the surgical aspect of this interesting case, has requested me to add a note from a physiological and medical point of view. Having had opportunities of watching the man during his illness, and believing

that the case presents features of importance, at the risk of some repetition I give the details as they came under my notice, in so far as they bear upon the question of cerebral localisation.

I saw the patient for the first time on December 30th, that is, eight days after the accident. He was perfectly well as regards general health, and all his organs and functions were normal. Intelligence was intact. Special senses normal. No pain in head. Pupils equal and normal. Fundus oculi natural. No apparent asymmetry of face. The patient said the right arm felt numb, but, on the most careful testing, there was no trace of objective loss of cutaneous or muscular sensibility. The movements of the right hand and fingers were only slightly clumsy, but otherwise no definite impairment in the actions of the upper extremity could be detected. The tendon reflexes and irritability of the muscles to percussion were the same on both sides. The movements of the right lower extremity were distinctly weak. The patient could walk, but with some difficulty. When lying in bed, the various actions of the right leg were performed but slowly, weakly, and unsteadily; the left was normal. Cutaneous and muscular sensibility of both lower limbs was natural. The knee-jerk was increased on the right side, but not to a great extent. Well-marked ankle-clonus right side, imperfect left. The exact position of the wound on the scalp was three-quarters of an inch to the left of the longitudinal line, and one inch behind a vertical line drawn through the external meatus of the ear.

During the next month the patient remained in exactly the same condition, with the addition, that he was occasionally seized with epileptiform attacks, as referred to by Mr. Davy. According to the statements of the nurse, these were all of the same kind. One of them I personally observed. Suddenly the right leg became rigid and extended; this rapidly spread throughout the whole of the right side; the patient then apparently lost consciousness, and the entire body, including the face, entered into a state of clonic convulsion; which lasted for a few minutes. After the attack, the patient was drowsy for an hour or so, and then regained his normal condition.

From this series of facts it was concluded that there was a lesion of the cortex cerebri on the left side, causing irritation and partial destruction of the area presiding over the movements of the right lower, and to a lesser degree over the right upper extremities. This was evidenced by the permanent paresis of the right leg and the slight weakness of the right hand, and by the epileptiform convulsions beginning in the

right lower extremity, extending throughout the side and ultimately over the entire body. From the position of the wound on the skull it was concluded that the wound of the brain was situated at the upper part of the ascending frontal convolution close to the longitudinal fissure. The diagnosis was confirmed in every respect by the operation, except that the injury to the grey matter was found further back than would have been anticipated from the position of the blow on the skull. The exact comparative size and locality of the destruction of brain substance caused by the manipulations necessary to remove the pieces of bone are indicated in the annexed diagrams. It will be seen that the cortex is absent throughout its entire thickness in a defined oval area at the upper part of the ascending parietal convolution, close to the longitudinal sulcus, and partly involving the postero-parietal lobule. Immediately below this a triangular portion of the corona radiata is destroyed. After death the parts marked white in the diagrams were strictly defined, occupied by firm cicatricial tissue, with normal cerebral matter in their immediate vicinity. The symptoms observed from the date of operation to the death of the patient from lung disease may be said to represent the effects caused by this local lesion of the brain. These were as follows: The patient was perfectly intelligent. The face and tongue were natural. The right upper extremity was slightly weaker than normal, but all its movements could be accurately performed. The right lower extremity was partially paralysed. It was not judged advisable to test locomotion; but when lying in bed, although the patient could flex and extend the limb, he did so slowly and feebly. It was difficult to say whether any special muscles were involved; there appeared to be general paresis of the whole limb. In the upper extremity the hand and fingers were chiefly affected, but even these only to a slight extent. There was nowhere any discoverable loss of sensibility. The tendon phenomena were increased in both lower extremities, chiefly on the right. The organs and functions of the body were otherwise normal.

The conclusion to be derived from the above observations is that a lesion of the cortex cerebri and subjacent corona radiata, strictly limited to the parts marked in the diagrams, is capable of causing marked but not complete paralysis of the lower, and slight paresis of the upper extremities of the opposite side, without any apparent disturbance of any other function or organ of the body.

A CASE OF CORTICAL PARALYSIS.

BY JOSEPH WIGLESWORTH, M.D. (LOND.)

Assistant Medical Officer, Rainhill Asylum.

ANN R., æt. 65, widow, was admitted into Rainhill Asylum, Sept. 8th, 1884, suffering from an attack of ordinary mania; this was her second attack, she having been under treatment in the same asylum for a similar mental affection, 5½ years previously. There was nothing at all peculiar in the character of the mental symptoms, which need not be again referred to. What concerns us here to note, is that she suffered from extensive necrosis of the skull, which was doubtless of syphilitic origin. At the time of her first admission, the inflammation of the cranial bones was actively in progress. We had on this, her second admission, to deal chiefly with the results of this disease, large portions of bone having come away, extensive irregularities of the upper surface of the cranium having thus been produced; over most of the depressions the skin was firmly cicatrised, but in some places small sores remained, covered with dry scabs.

About the middle of February, 1885, patient was laid up with a mild attack of facial erysipelas; on recovery from which (20th inst.) it was noticed that pulsation existed at the bottom of one of the cranial depressions. Though no bone was found, it was clear that a small portion had come away, for there was now a small circular perforation, somewhat less than a shilling in size, through which the dura mater protruded, partially covered with granulations. The perforation was situated a little to the left of the middle line, and about the centre of a line drawn from the forehead to the occiput; the irregularity in the surface of the cranium prevented more accurate measurements. On the evening of the 25th inst. the nurse had just completed taking patient's temperature (102·2°), when she suddenly took an epileptiform fit, which was shortly afterwards repeated; in both these fits the head and eyes were first turned to the right, then the right arm and hand were drawn up and convulsed, and shortly afterwards the left side was similarly convulsed. A dose of chloral was given, and the convulsions were not repeated. From this date, however, patient gradually became dull and heavy, lying quiet, making

no attempt to answer questions and not appearing to understand them [she had a good deal of reasoning power before this attack came on]; she also seemed distinctly deaf. On March 8th (eleven days after the convulsions) her right arm was found to be partially paralysed, exhibiting also some amount of rigidity: six days subsequently (14th inst.), the right arm had become completely flaccid and helpless, and the paralysis had spread to the right leg, which, though not absolutely devoid of movement, exhibited great defect of this. As the dura mater was now swollen and tense in the perforated area, it was incised, and about half a drachm of thick creamy pus escaped. No improvement, however, resulted in the symptoms. The cerebral substance now presented itself at the cranial opening in the form of a globular, pulsating mass.

The patient gradually became weaker, presenting ever increasing mental hebetude; she did not speak at all, and appeared hardly to understand anything that was said to her. Pupils 3 mm., equal, regular, acted well to light and to accommodation. The right arm remained perfectly paralysed and flaccid, but the paralysis of the right leg was never quite complete.

The patient gradually became more comatose, and died on March 24th, twenty-seven days after the access of the convulsions.

Autopsy (seventeen hours after death):

Cranium.—Skull-cap presented numerous irregular depressions from localised losses of substance, in both frontal and parietal regions; in parietal region, a little to the left of middle line, an opening about the size of a shilling, through which brain-tissue protruded. Dura-mater somewhat abnormally adherent to skull: a little thin recent blood clot smeared over inner surface of dura-mater at anterior part of left frontal lobe. Very little fluid in subdural space. Pia-mater over left frontal lobe somewhat hyperæmic, but both here, and over the right hemisphere, it was thin, and stripped readily. Slight opacity of arachnoid here and there in right hemisphere. Longitudinal sinus contained soft recent black clot, and lateral sinuses distinct ante-mortem, partially decolorised thrombi, which were not, however, distinctly adherent to the walls of the vessels. Inner surface of dura-mater rather firmly adherent to arachnoid over a good portion of left parietal lobe, and also, although to a less extent, over left temporo-sphenoidal lobe; on separating these membranes, thick greenish pus escaped from a rent, situated near the upper end of the left ascending parietal convolution. The portion of disorganised brain-tissue which protruded through the cranial perforation, was found to consist of the upper three-fourths of

an inch of the left ascending parietal convolution, a portion of the upper part of the paracentral lobule, and a very small portion of the adjoining part of the ascending frontal gyrus. The pus which escaped was found to communicate with an abscess cavity about the size of a pigeon's egg, and containing nearly an ounce of pus, which was situated in the centrum ovale, extending towards the lateral ventricle, but not reaching this; it had hollowed out the greater part of the ascending parietal gyrus, which collapsed into it; this gyrus, however, retained its cortex (although this was superficially softened), with the exception of the upper part, which, as above mentioned, was disorganised, and protruded through the perforation. There was also a small abscess cavity in the left angular gyrus, and some superficial softening of the cortex of the left first and second temporo-sphenoidal gyri, which were smeared with pus, and a vein of some size running along the upper margin of this lobe was plugged with an ante-mortem thrombus. Ventricles not obviously dilated. Basal ganglia perfectly normal, as indeed was the right hemisphere; cerebellum, pons, and medulla also normal.

Brain (immediately after removal) = 1185 grammes.

Right hemisphere = 505 grammes.

Left hemisphere = 524 grammes (both stripped of membranes).

Cerebellum = 123. Pons = 18. Med. Obl. = 9.

Basal vessels healthy.

Thoracic and *Abdominal* viscera normal, with the exception of partial consolidation of the posterior half of the lower lobe of the left lung.

Remarks.—Though the lesions in this case were a little diffused, the case is nevertheless of interest, in that, an irritative, and subsequently destructive lesion of the left motor area was associated first with convulsions, and afterwards with paralysis of the limbs of the right side of the body. The left ascending parietal convolution was practically completely functionless, for though the cortex was not wholly destroyed, the fibres proceeding therefrom seemed quite to be so, and the lesion extended on to the median aspect, involving a portion of the paracentral lobule. The paralysis of the opposite limbs hence finds a ready explanation, whilst the convulsions to which the paralysis was consecutive, commencing on the right side, and subsequently spreading to the left, followed the course noted in electrical stimulation of the cortex. It is perhaps legitimate to associate the superficial softening of the left first and second temporo-sphenoidal gyri with the deafness from which the patient seemed to suffer; for though this symptom was difficult to disentangle from the general hebetude, it was remarked during life before deep coma supervened.

DISTRIBUTION OF ANÆSTHESIA AFTER DIVISION OF THE MEDIAN NERVE.

BY C. W. SUCKLING, M.D. (LOND.), M.R.C.P.

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In the April number of "BRAIN" for last year there is a paper by Dr. James Ross upon the distribution of anæsthesia in cases of disease of the branches and roots of the Brachial Plexus. The following case, at present under my care, supports several statements in that paper. Krause's account of the distribution of the digital branches of the nerves of the hands is fully confirmed, except that in my case there was complete anæsthesia of the dorsum of the ungual phalanx of the thumb.

The anæsthesia was complete for some time after the accident, and was far more extensive than in the cases observed by Létiévant.

The patient, a medical student, was reaching a bottle of soda-water out of its case, when the bottle burst in his hand, severely cutting his right wrist in front and in the middle line. This occurred on the 25th of September last.

At the time of the accident he perceived, as it were, a great rush of blood into the hand, which felt as though it was being greatly distended, and became cold and numb.

During the dressing of the wound, and every time the pad was used to swab up the blood so as to look for a divided nerve or artery, the pain was most acute, which was probably due to the pad coming in contact with the divided nerve.

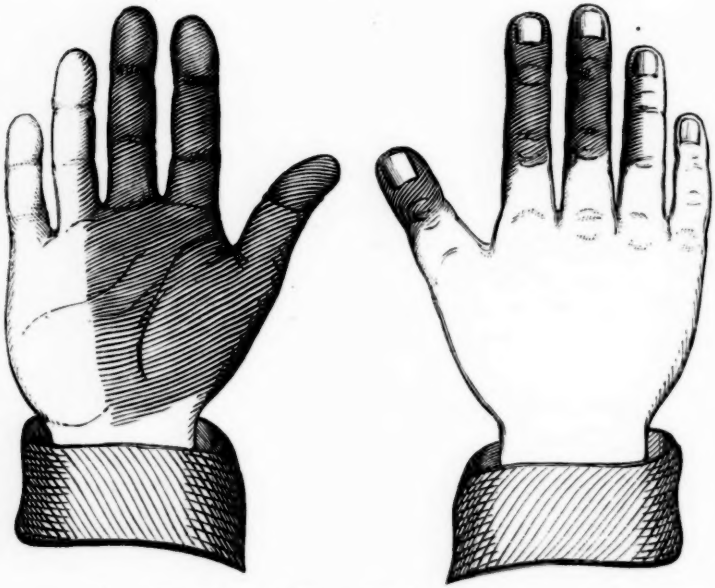
The nerve was not seen during the treatment of the wound. The hand was put in splints for three or four days; and on their removal he found that the muscles of the hand supplied by the median nerve were paralysed, and that there was total anæsthesia in the area of distribution of its sensory branches.

Three days after the accident there was total anæsthesia of the radial half of the palm and thenar eminence, the anæsthetic area being sharply defined and not gradually shading off; it extended in the palm to a line contiguous with the axis of the ring-finger.

There was also complete anæsthesia of the palmar surfaces of the thumb, index, middle and radial half of ring-finger.

On the dorsum of the hand there was total anæsthesia of the ungual phalanx of the thumb and of the second and third phalanges of the forefinger and middle finger, and of the radial halves of the same phalanges of the ring-finger.

A few days after the accident he began to feel sharp shooting-pains in the hand and fingers, which would come on suddenly and as suddenly disappear. These pains frequently prevented sleep, but after lasting for about a month they gradually ceased.



FIGS. 1 and 2.—THREE DAYS AFTER ACCIDENT.

The Shaded portion represents complete Anæsthesia.

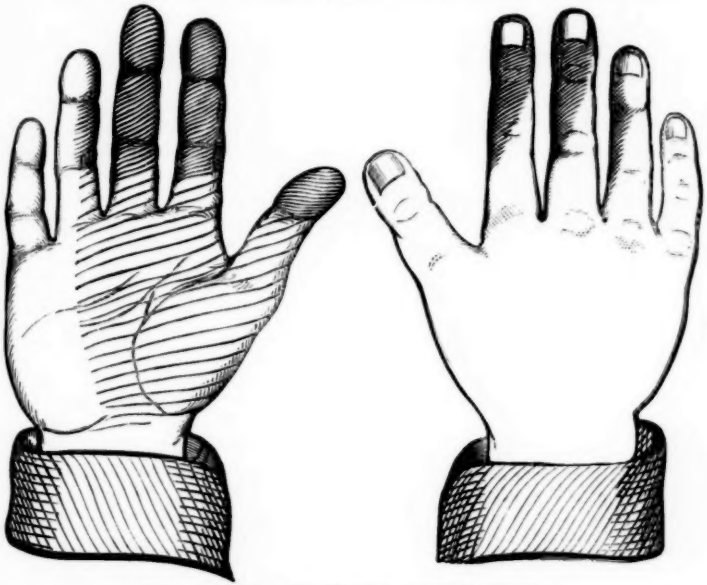
When he attempted to move the fingers he was always troubled with a sharp pain of a tearing character, which would at times extend into the forearm; this was especially acute on moving the index-finger. This pain also gradually ceased.

Since the accident there has been a great diminution of temperature in the three and a half fingers, and during the cold weather, he had great difficulty in keeping them warm. After exercise he frequently noticed the little and half the ring-finger and the inner part of the hand to be covered with

perspiration, while the rest of the palmar surface of the hand was quite dry.

At the present time (February), nearly five months after the accident, there is a scar in front of the right wrist passing obliquely across the tendon of the flexor, carpi radialis and median nerve. The scar is very sensitive when touched. A small bulla, containing blood, is seen on the inner side of the terminal phalanx of the index-finger.

No changes in the nails are to be observed. He has re-



FIGS. 3 and 4.—FIVE MONTHS AFTER ACCIDENT.

The portion thickly Shaded represents the Anæsthesia; the thinly Shaded portion Hyperæsthesia.

covered power to a considerable extent, being able to write with the right hand; but the grasp is much weaker than on the left side.

There is wasting of the thenar eminence on the right side. The circumference of the base of the right thumb being five inches, that of the left, five and a half. The response of the muscles of the hand supplied by the median is much diminished to faradism. To galvanism the response is increased, the contractions being slow and prolonged, the anodic closure contraction predominating.

The distribution of the anæsthesia at the present time, nearly five months after the accident, is as follows. The area of the right palm previously anæsthetic is now everywhere hyperæsthetic, a slight touch causing pain, the pain radiating into the fingers.

The hyperæsthesia extends over the palmar surface of the first phalanx of the thumb and encroaches on the first phalanges of the forefinger, middle finger, and ring finger.

The skin of the rest of the phalanges are still anæsthetic on slight pressure, but hyperæsthetic on deep pressure, the pain radiating into the fingers.

On the dorsum of the hand the following changes have occurred:

The anæsthesia previously present on the dorsal aspect of the ungual phalanx of the thumb has disappeared. The only alteration in the other fingers is that the anæsthesia of the middle phalanges of the fore and middle fingers extends now only over the radial half of the phalanges, the ulnar half being normal.

A CASE ILLUSTRATING THE CONDITION OF THE NERVOUS SYSTEM AFTER AMPUTATION OF AN EXTREMITY.

BY WILLIAM DUDLEY, M.B. (LOND.),

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THE published cases in which have been described lesions of the nervous system as the results of amputation, are so few, that the following seems worthy of record.

Adolph K——, 39, married, was admitted into the West Riding Asylum, Feb. 28th, 1885.

History (obtained on admission).—Patient, who is a Pole, according to his own account, lost his left leg, through bullet-wounds received in 1864. During the past three years he is said to have been excitable, and to have behaved frequently in a brutal manner towards his wife and his employés. About five weeks ago he was thrown from a trap, and sustained a fracture of the right leg. Symptoms of insanity were first observed while he was confined to bed with the broken leg.

On admission, there was an ununited fracture of the right leg. The left leg had been amputated at the junction of the upper and middle third of the thigh.

The case was an ordinary one of general paralysis, and death from exhaustion occurred on March 25th, the fatal termination being hastened by suppuration at the seat of fracture, and by large bed-sores.

At the autopsy, 26 hours after death, the fractured ends were found necrosed; the surrounding tissues were infiltrated with pus; there was fibroid induration of the lungs, and deep congestion of the lower lobes; there was slight atheroma of the aorta; the kidneys were slightly cirrhosed.

The brain showed the appearances ordinarily met with in a moderately marked advanced stage of general paralysis, viz. atrophy, excess of serum (five ounces being collected), opaque, thickened and tough membranes, with cortical adhesions of small superficial extent and slight depth.

The ascending parietal convolution and the postero-parietal lobule of the right side were smaller than those of the left. The right ascending frontal was crossed at 1½ in. from the longitudinal fissure by a deep sulcus. Its knee was much smaller than that of the opposite side. A naked-eye examina-

tion revealed nothing abnormal in the basal ganglia, the cerebellum, the pons and medulla.

In sections taken from the ascending frontal, the ascending parietal, and the postero-parietal lobule of the right side the cortex is seen to be of average depth, and the different layers of cells to be distinct. The nerve-cells do not appear to be diminished either in size or in number; some of them show pigmentary degeneration, and the ground substance is more deeply stained than occurs in the normal condition, these changes being similar to those usually met with in general paralysis, and may probably have no relation to the loss of the left lower extremity.

Spinal Cord.—In the cervical and dorsal regions there is no appreciable difference in the grey or white matter of opposite sides. The cells are equally numerous, and have undergone degeneration to a similar extent. The white matter is healthy.

In the dorso-lumbar region no decided difference in the two sides can be detected. At the thickest part of the lumbar enlargement a difference in size of the anterior cornua is observable, the right being the larger. Microscopically, the cells of the right side are found to be a little more numerous than those of the left.

About one inch from the lower extremity of the cord, a difference in the sectional area of the grey matter of the two sides is obvious, the transverse measurement of the right anterior cornu being to that of the left in the proportion of 3 to 2. From this level downwards the difference diminishes, but can still be seen even when the diameter of the cord does not exceed a line. The right posterior cornu is very slightly larger than the left.

At the level at which the difference in size of the anterior cornua is most marked, a still more obvious difference in the number and size of the cells is seen upon microscopic examination, about 15 to 18 fairly plump cells being present in a transverse section on the left side, whereas there are about 40 to 45 on the right; moreover, the majority of those on the left side are distinctly smaller than those on the right, so that if the 30 largest cells in both cornua be taken, about 24 or 25 will belong to the right side. The antero-lateral and postero-lateral groups are those in which the difference is most evident. No morbid change is detected in the white matter. Near the lower extremity of the cord it is ragged and ill-stained, probably owing to its having been sodden with serum.

Throughout the cord, side by side with cells which appear perfectly healthy, are numerous cells which have undergone

pigmentary degeneration. Some of these appear to be of normal size, and show a distinct cell-wall with numerous well-stained processes; but in place of healthy protoplasm, which readily takes up most staining reagents, the greater part of the interior of the cell is occupied by yellow granular pigment, which does not stain at all, or but very faintly. Other cells have a rounded outline, and are frequently pear-shaped, and appear swollen, from the presence of a mass of pigment, while the very small amount of healthy protoplasm which remains appears as if pushed away to the narrow end of the cell, from which a well-stained process is usually seen to extend, the other processes being faintly stained, or invisible. Other cells show a cell-wall enclosing nothing but coarsely granular pigment, and have only few and feebly-stained processes. These cells are generally, but not always, smaller than the more healthy cells in their immediate neighbourhood. Finally, there are found masses of pigment, with neither protoplasm nor processes, and these are in all probability the remains of cells which have entirely lost their function. These extremely degenerate elements are much more numerous in the lumbar region, and on the left side; but judging from their number in this situation, it is probable that here some cells have disappeared entirely, so as to leave no trace behind.

Within the cord, the fibres of the anterior nerve-roots seem to be more numerous on the right than on the left side, at the level at which atrophy is well-marked. Among the fibres of the left side are here and there to be seen axis cylinders, represented by a line of dots or by a row of short irregular broken lines, and deeply-stained granular masses are also found in this situation, which probably represent degenerate fibres.

Transverse section of the left anterior crural nerve show bundles of very fine nerve-fibres scattered irregularly between healthy fibres. These fine fibres are seldom isolated. They occur in groups containing from two or three up to thirty or forty, and the groups appear, on section, of the most varied shape. Some of the fasciculi contain more of the fine fibres than others do; none appear to be quite free. The fine fibres stain much more deeply than the healthy ones. Their diminished size appears to be due mainly to a much-attenuated medullary sheath. Their diameter varies considerably, but the majority are about one-third or one-fourth that of the healthy fibres. A large amount of adipose tissue exists between the fasciculi. It is to be regretted that the left anterior crural nerve was the only one preserved.

A CASE OF ACUTE MANIA, IN WHICH THERE WAS COMPLETE REMISSION OF THE SYMPTOMS DURING A TEMPORARY PLUGGING OF THE SINUSES.

BY T. BULKELEY HYSLOP,

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M. S., æt. 41, married, housewife, admitted into the West Riding Asylum, July 1882, suffering from an attack of acute mania.

The attack began a week previous to admission, to account for which there was no special illness, shock or trouble.

About sixteen months before, she had been confined, and had suckled her child up to the date of the present illness. She was of good moral character, and a steady hard-working woman.

She is said to have suffered from depression and nervousness for some weeks before admission. She has had ten children, seven of whom are alive and well. After her last confinement, catamenia were regular.

Family History.—A maternal uncle died in this asylum, and her grandfather died insane. No history of epilepsy, apoplexy, or phthisis.

On admission, the patient was in a state of acute maniacal excitement; she struggled incessantly, and was quite incoherent and hoarse from continuous raving. She was the subject of delusions, stating that there was war in the village where she lived, and wildly shouted, "John the Baptist is my son." She appeared alarmed at times, and would cry out that the children in the house were being killed.

Her physical condition was fairly good, pupils equal. The right mamma was larger than the left.

For some weeks after admission the patient continued in the same state of restlessness and maniacal excitement, causing her to become pale, anæmic, and much reduced.

August 11*th.*—There was noticed an appearance of redness and fulness of the left ear, which was found to be hot, and suggestive of commencing hæmatoma auris.

September 12*th.*—The left ear commenced to shrink, but

hæmatoma of the right ear had developed. The patient was more excited than ever, continually abusing those around her, wrestling with the attendants, and violent to every one that approached her.

January 15*th.*—A marked change came over the patient. She became suddenly quiet and composed, talking in a rational manner, and showing no sign of mental weakness. She was slightly feverish, however, with a throbbing pulse, and there was noticed in the region of the parotid gland a distinct swelling and tenderness.

16*th.*—The parotid swelling was found to have subsided, but over the mastoid cells on the right side a painless tumefaction had appeared. There was also in the right orbital region a general puffy and œdematous condition. She complained of occipital headache.

18*th.*—The swelling in the orbital region was considerably diminished, and the patient still continued quiet and rational. She complained, however, of pain in her throat, which was found to be slightly congested.

February 2*nd.*—The œdematous condition was found to have quite subsided, and the tumefaction over the mastoid cells had completely disappeared, whilst the patient herself had just as suddenly relapsed into her old ways of disorderly excitement. She was garrulous and abusive as formerly, and in much the same state of persistent excitement she has continued up to the present time, taking her food badly and sleeping little, whilst her physical condition has become much reduced.

It is of interest to note in this case, that the sudden remission of the maniacal symptoms coincided in point of time with the appearance of the œdema of the orbital region, and over the mastoid cells; whilst the disappearance of this œdema was attended with just as sudden a relapse into the maniacal state.

Critical Digests.

RECENT OBSERVATIONS ON DEGENERATION, AND ON NERVE TRACTS IN THE SPINAL CORD. A CRITICAL ACCOUNT.

BY J. N. LANGLEY, M.A., F.R.S.

Fellow and Lecturer of Trinity College, Cambridge.

1. ***Langley and Sherrington.*** "Secondary Degeneration of Nerve Tracts following removal of the Cortex of the Cerebrum of the Dog." 'Journal of Physiology,' v. p. 49. 1884.
2. ***Pitres.*** "Recherches anatomo-cliniques sur les scléroses bilatérales de la moelle épinière consécutives à des lésions unilatérales du cerveau." 'Arch. de Physiologie normale et pathologique,' p. 142. 1884.
3. ***Löwenthal.*** "Des Dégénérations secondaires de la moelle épinière consécutives aux lésions expérimentales médullaires et corticales." 'Dissertation.' Genève (Schuchardt), 1885.
4. ***Sherrington.*** "On Secondary and Tertiary Degenerations in the Spinal Cord of the Dog." 'Journal of Physiology,' vi., p. 176. 1885.
5. ***Homén.*** "Experimenteller Beitrag zur Pathologie und pathologischen Anatomie des Rückenmarkes." 'Fortschritte der Medicin' (No. 9, May), p. 267. 1885.

THE broad outlines of our knowledge of the nerve tracts of the spinal cord, we owe very largely to the observations on degeneration, made by Turck[1] in 1851 to 1853, and by Bouchard[2] in 1866. Since 1866 there have been many observations confirming in the main the results of Bouchard, but new results have come slowly and sparsely. The principal additional facts are perhaps those noted by Schiefferdecker[3] in 1865, and by Singer[4] in 1881. The general acceptance of these results is due chiefly to the work of Flechsig[5] in 1876, on the times at which the various nerve-fibres in the spinal cord acquire their medulla. The degeneration

[1] Türck, 'Sitzungsb. d. Wiener Akad.,' Bd. vi. p. 288, 1851; Bd. xi. p. 93, 1853; also in Bd. xiv. p. 329, 1855.

[2] Bouchard, 'Archives Générales de Médecine,' vi. ser. t. vii. pp. 272, 441, 561; t. viii. p. 273 (1866).

[3] Schiefferdecker, 'Virchow's Archiv,' Bd. 67, p. 542, (1876).

[4] Singer, 'Sitzungsb. d. Wiener Akad.' Bd. 83, p. 390, (1881).

[5] Flechsig, 'Die Leitungsbahnen im Gehirn u. Rückenmark des Menschen,' (Leipzig) 1876.

method and Flechsig's method gave similar results, and the mass of detailed information brought forward by Flechsig produced a belief not accorded to the previous observations on degeneration. Moreover, Flechsig's description was much more complete than that of his predecessors.

But there still remain many points undetermined. I propose in the following paper to discuss these points on the basis of the observations made in 1884 and in 1885. In doing so it will be necessary, for the sake of clearness, to refer to many of the earlier observations.

We will first take the changes which can be observed in secondary degeneration, since some of the discrepancies between the accounts of different writers are due to the different methods employed, and to the different times after the lesion at which the cord is examined.

The characters of secondary degeneration are the same, whether it is caused by lesion of the spinal cord or of the brain. We will, then, refer to cases of secondary degeneration produced by either cause. And for convenience we will take separately the changes observed according as the spinal cord is, (1) cut across in the fresh state and examined straightway; or (2) hardened in potassium bichromate or other similar agent and then cut across and examined; or (3) hardened, and sections stained and examined with the microscope.

1. *Spinal Cord examined in the fresh state.*—In a transverse section of the fresh cord, the degenerated area has a more milky-white appearance than the rest of the white substance; this is apparently due to a change in the medulla of the nerve fibres. According to Löwenthal, this change can be seen as the result of section of the cord, or of injury to the motor area of the brain, at all times between three and twelve weeks after the lesion. His observations on the result of section of the cord do not extend beyond these limits. He has made one observation at an earlier date on the effect of cortical brain lesion. The cord was examined fifteen days after the injury. A milky-white spot was present in the lateral pyramidal tract in the cervical region; whether it was present or not in the rest of the cord he does not mention.

Sherrington was unable to observe in the dog the "milky" degenerative change in the lateral pyramidal tract, after injury to the motor area of the cortex, earlier than the fifth week. In three to four months, according to Sherrington, the spot becomes less white, and then greyish, at the same time acquiring a more transparent and gelatinous look. This is the state in which it has most commonly been described in the fresh cord.[1] Finally, it becomes unrecognisable in the fresh cord, although after the cord is hardened it may still readily be seen.

2. *Spinal Cord hardened in Potassium Bichromate.*—The degenerated area in this case appears as a yellowish spot of lighter tint than

[1] For a *résumé* of the descriptions which have been given of the appearance of the area of degeneration in the fresh cord, cf. Löwenthal, op. cit. (3), p. 84.

that of the rest of the cord. Singer[1] observed this in the cerebellar tracts, and in the columns of Goll of the dog, throughout the cord, twelve days after section in the lower dorsal region.

Löwenthal describes the descending degeneration as being thus visible, though not very distinctly, twenty-one days after hemisection of the cord of the dog in the cervical region, and as being much more obvious three months after the injury.

Sherrington finds that after destruction of the motor area of the cortex, the yellowish spot of degeneration is visible in nine days from the 1st cervical to the 2nd lumbar nerve-roots; but is, up to a limit of three to six months, the more distinct the longer the degenerative changes have been going on. Towards the end of this time it becomes smaller. In degenerations of longer date than six months the change is less distinct. It is not quite certain whether long-standing secondary degenerations ever become quite unrecognisable in the hardened cord on macroscopic investigation; they are, however, in their later stages more easily recognised under the microscope.

3. *Examination of sections of the Spinal Cord under the Microscope.*—Homén[2] in 1883 found in the dog that, after hemisection of the cord in the lower dorsal region, degenerating fibres were present in the cord *seven* days after the injury both above and below the place of section. In these fibres he found, as a rule, the neural axis (axis cylinder) only affected. In eleven days the number of fibres with altered medulla was greatly increased. In twenty-one days the neuroglia cells were more conspicuous, and some of the nerve-fibres had disappeared. In twenty-nine days the general connective tissue-substance between the nerve-fibres showed signs of alteration.

Homén in his more recent paper (5) again considers the time after hemisection of the cord at which degeneration can first be made out, and the nature of the degenerative changes. He finds that the degenerative changes do not begin at the same time in the various nerve tracts. In *three* days altered fibres can be seen in the posterior columns,[3] and can just be made out in the lateral pyramidal tract; in five days the degeneration in the lateral pyramidal tract is distinct; in seven days altered fibres are present in the cerebellar tract. During the first few days of the degenerative change, the number of affected fibres rapidly increases; how long this increase goes on is not stated.

For detecting the early stages of degeneration Homén recommends staining with acid-fuchsin. As we know from Schieffer-

[1] Singer, op. cit.

[2] Homén, 'Comptes Rendus,' No. 23, 1883. Schiefferdecker (op. cit.) did not find any altered fibres seven days after section of the cord of the dog in the lower dorsal region; fourteen days after section altered fibres were present, the medulla of the nerves stained slightly, the neural axis stained less than normal. Sherrington describes altered nerve-fibres in the cord nine days after cortical lesion in the dog.

[3] He finds that the change is distinct through the whole of posterior column above the place of section.

decker and others, the first change seen in the nerve-fibres is that the neural axis becomes somewhat swollen and granular, and stains less than normal with carmine. Homén finds that acid-fuchsin stains the degenerating neural axis much more deeply than the normal, and of a dark-red colour; so that the degenerating fibres are much more obvious than in specimens stained with carmine.

As the myelin of the nerve-fibres becomes affected, it stains slightly with carmine, and becomes with difficulty or not at all distinguishable from the neural axis. When acid-fuchsin, or hæmatoxylin after Weigert's method, is used, the inner part of the myelin first blends with the neural axis, the outer part staining as before—bright red with acid-fuchsin, black with hæmatoxylin—later, in three to eight weeks in different fibres, the outer part of the myelin sheath also blends with the axis, forming a mass which is nearly homogeneous, and stains but little. As this is going on, the fibres split up, and the separated swollen portions form colloid bodies, which are gradually absorbed.

Thus it appears that the neural axis is first affected, then the medulla, and lastly the connective tissue. The hypertrophy of the connective tissue is probably due to the irritative action of the substance of the dead and disintegrating nerves.

That the connective tissue is only secondarily affected has been laid stress on by nearly all observers.[1] When a few fibres only degenerate, the connective tissue may be unaffected: in such cases, degeneration can only be seen during the process of breaking down of the nerve-fibres; if the fibres are scattered, the degeneration can only be seen under the microscope.

In the early stages of connective tissue change, Deiters' cells are fairly numerous and conspicuous, and there are many granular cells and leucocytes; according to Bouchard, the blood-vessels are atheromatous. In the later stages these characters largely disappear; the connective tissue remains in abnormal amount, and is usually said to consist of a network of fine fibres. Sherrington and myself found it to consist almost wholly of fine semi-elastic fibres, not anastomosing with one another, and staining with carmine. We considered, too, that the connective-tissue "scar" diminished in time in staining power, and hence became more difficult to recognise.

The following may be taken as a rough summary of the chief points connected with the observation of secondary degeneration, although no doubt several of the statements are open to discussion. Secondary degeneration is best observed with the microscope four to five weeks after the injury. The degeneration can be made out near the lesion in a week or less. It spreads fairly rapidly—probably in less than twenty-four hours—along the whole course of the nerve-fibres. It can be earliest seen by the alteration in the neural axis, a day or two later by the change in the myelin sheath also. In one to two weeks it can be observed by the eye in the cord hardened in

[1] *e.g.* Schiefferdecker, op. cit. Kahler and Pick, 'Arch. f. Psychiatrie,' Bd. x. pp. 179 and 279 (1880); Schultze, 'Arch. f. Psychiatrie,' Bd. xiv. p. 358 (1883).

potassium bichromate. In about three weeks[1] it can be seen by the eye in the fresh cord. The neuroglia now begins to increase, and the nerve-fibres to be absorbed, although probably none disappear for some weeks. In consequence of these changes, the degeneration is, from the sixth week onwards, more and more noted under the microscope by the change in the neuroglia, and less and less by the presence of altered nerve-fibres. In the lateral pyramidal tract altered nerve-fibres are seen for three to four months. The length of time after the lesion during which the area of degeneration can be detected by the "scar," varies with the number of fibres which have degenerated; in the lateral pyramidal tract the scar can be seen for a year, and probably for much longer. The degeneration can be seen in the fresh cord for four to five months; during the last six weeks or so of this time, it changes from milky-white to greyish. Possibly in the cerebellar tract the change takes place earlier. In time (? six months) no change can be seen in the fresh cord. In the cord hardened in potassium bichromate, the area of degeneration becomes more distinct for four to six months; later than this it becomes less and less distinct; in the lateral pyramidal tract it may be seen for about a year. In its earlier stage, the area of degeneration may appear larger than it really is, in consequence of a spreading out of the connective tissue change; in its later it becomes smaller, in consequence of a retraction (? partial disappearance) of the connective tissue.

Whilst all observers have paid considerable attention to the time after the lesion at which secondary degeneration makes its appearance, comparatively little attention has been paid to the time at which the degeneration of the nerve fibres is complete. But this is a point of some importance. According to Löwenthal, the degenerating areas in the region of the lateral pyramidal tract, which result from sections of the cord in the cervical region, consist chiefly of altered fibres three months after the section. He finds, too, that the number of altered fibres is greater in twelve weeks than in six, and greater in six weeks than in three. Sherrington describes a few altered fibres as being present in the lateral pyramidal tract seven months after cortical lesion. But from the descriptions of most observers, we should gather that some fibres disappear altogether in about a month. Homén, in three weeks, finds spaces from which fibres have disappeared; but it is not clear whether the fibres had been absorbed from these spaces, or simply dropped out of the section. If fibres do disappear so early, and if Löwenthal's statement regarding the increase in the number of altered fibres for two to three months after the lesion is correct, it follows that some fibres have disappeared, whilst others have scarcely begun to degenerate. This seems extremely unlikely, if all the fibres are undergoing true secondary degeneration.

It seemed possible that there might be some differences between descending and ascending degeneration in these respects; but I cannot find that any one who deals with both degenerations points out any difference. The only clear statement as to the time of disappearance of nerve-fibres in ascending degeneration which I have been able to find is given by Singer. According to

[1] In man it is often difficult to observe any change in the fresh cord; possibly this may be on account of the time which elapses before the cord is examined.

him, in *young* dogs after section of the cord in the dorsal region, the nerve-fibres of the cerebellar tract and of the medial part of the posterior column completely disappear in four to five weeks. In the adult dog, the process is slower; unfortunately he does not say how much slower. There seems to me, however, to be *primâ facie* ground for thinking that, at any rate in the pyramidal tract, some fibres do not begin to degenerate until several weeks after section of the cord.

Turning now to the nerve-tracts in the cord, we may first consider—

The Pyramidal Tracts.—Flechsig mentions that in the lateral pyramidal tracts there are a certain number of fibres which do not come from the opposite pyramid. But in addition to these fibres, it is probable that the lateral pyramidal tract, as described by Flechsig, contains many other fibres which do not belong to this tract.

Bouchard in 1866 noticed that in man, the descending degeneration in the lateral column was considerably greater after injury to the medulla than after injury higher up in the brain.

Bouchard[1] describes the area of degeneration which appears in the cord in the posterior part of the lateral column after injury to the medulla, as extending to the surface. Since the cerebellar tract occupies the periphery of this part of the cord, and does not undergo descending degeneration, Bouchard's statement regarding the peripheral strip probably rests on a misinterpretation of the appearance. According to Schiefferdecker, the sclerosis may extend beyond the area of the degenerated fibres; to this the sclerosis seen by Bouchard at the periphery of the cord was probably due.

The fibres which degenerated after injury to the medulla, but not after injury to the brain, were situated on the lateral border of the pyramidal tract. These fibres he called "fibres commissurales antérieures longues." This observation has passed with little or no notice.

A somewhat similar difference, however, is described by Löwenthal, as occurring in the dog, after section of the cord in the cervical region and after removal of the motor area of the cortex. According to Löwenthal, the long commissural fibres lie between the cerebellar tract and the lateral pyramidal tract; they are chiefly large fibres; whereas, as we know, the pyramidal tract fibres, like those of the pyramid, are chiefly moderate-sized and small. Löwenthal points out also that, after section of the cord in the cervical region, the degenerated area in the lateral column has a fairly sharp boundary on its lateral edge. In degeneration after removal of the cortex of the brain, this is certainly not the case, the degeneration becomes quite diffuse as it approaches the cerebellar tract.

Sherrington also has come to the conclusion that, at any rate in the cervical region of the cord, the real pyramidal tract occupies a part only of the pyramidal tract as figured by Flechsig. This conclusion is, however, based upon a comparison of the area of

[1] Bouchard, op. cit. t. vi. p. 570.

degeneration which he obtains in the dog, with the area of non-medullated fibres seen in the developing cord of the ape, and with the area of the pyramidal tract as given by Flechsig in the cord of man. The basis is somewhat uncertain. Proceeding, however, in this way, he concludes that the ventral and the latero-dorsal parts of Flechsig's pyramidal tract do not degenerate from cortical lesions, and hence are not continuations of the pyramid fibres.

On the whole, it seems probable that the pyramidal tract is much smaller than that given by Flechsig, that it does not form a compact bundle of fibres, but on its anterior (ventral), lateral and posterior (dorsal) borders, it becomes diffuse, its fibres being mixed with other fibres, many of which, especially on its lateral border, degenerate on section of the spinal cord. In its inner (mesial) side its fibres, as described by Flechsig, become mixed with the small fibres of the "boundary layer." It will be noticed that, as far as Löwenthal's experiments extend, the "anterior long commissural fibres" might be "commissural"[1] fibres of the cord. From Bouchard, however, we should conclude that they run from the medulla to the various segments of the cord, and hence we might call it the descending medullary tract.

Bilateral Degeneration of Lateral Pyramidal Tract in consequence of Unilateral Cortical Injury.—It has been known for some time, that in man an injury confined to one side of the cord, might cause a degeneration in the lateral pyramidal tract of the opposite as well as in that of the same side of the cord. Charcot[2] suggested as an explanation of this, that part of the lateral pyramidal tract crossed in the anterior commissure, especially in the upper dorsal region, to the lateral tract of the opposite side. Thus the fibres would first cross in the pyramidal decussation, and then again in the anterior commissure.

A case of bilateral degeneration in the cord of the dog, as the result of unilateral cortical injury, was described in 1880 by François-Franck and Pitres.[3]

Moeli[4] in 1883 found after unilateral injury to the mid-brain in dogs, that besides the degeneration of the pyramidal tract of the opposite side there was a degeneration of a few scattered nerve-fibres in the pyramidal tract of the same side.

An instance in the ape has also been described by Schäfer[5] (1883); the examination was made seven months after the cortical injury. In the cervical region of the cord there was sclerosis in both lateral pyramidal tracts, the area of the two sides being about equal; but the sclerosis was less in intensity on the same side as the lesion, and on this side it could not be traced beyond the cervical region.

[1] Cf. p. 106.

[2] Charcot, 'Leçons sur les localisations dans les maladies du cerveau et de la moelle, recueillies par MM. Bourneville et Brissaud,' p. 252 (Paris, 1876–1880).

[3] Francois-Franck et Pitres, 'Progrès Méd.' p. 146 (1880).

[4] Moeli, 'Arch. f. Psychiatrie,' xiv. p. 173 (1883).

[5] Schäfer, 'Jour. of Physiology,' vol. iv. p. 324 (1883).

The bilateral degeneration of the pyramidal tract has received a good deal of attention from recent observers. In no case apparently does unilateral brain-injury cause degeneration of the opposite pyramid,[1] so that whilst one pyramid only degenerates, there is degeneration in the region of both the lateral pyramidal tracts in the cord. We will, for lack of a better term, call the degeneration which occurs on the side of the cord opposite to the sound pyramid the *re-crossed degeneration*.[2]

Pitres examined the spinal cord in forty cases of unilateral brain-injury in man. In ten of these there was on both sides sclerosis of the lateral pyramidal tracts. In some cases this could only be made out on microscopical examination. In six out of the ten cases the sclerosis was symmetrical and equal on the two sides, and on both sides it occupied a larger area, chiefly in the ventral direction, than that occupied when sclerosis occurred on one side only. In the remaining four cases the re-crossed degeneration was of less intensity than that of the opposite side. According to Pitres, the re-crossed degeneration stretches throughout the cord, reaching the surface of the cord in the lumbar region. No relation could be made out between the presence or absence of a re-crossed degeneration, and the presence or absence of a degeneration in the medial part of the anterior column (the direct pyramidal tract, Türck's column). In six cases the direct pyramidal tracts showed some sclerosis; this, whilst varying in amount, was in all cases slight, and in one case only could it be traced in the lower dorsal and in the lumbar region. In three cases there was sclerosis in *both* direct pyramidal tracts.[3]

Pitres considers the re-crossed degeneration to be due to the degeneration of fibres coming from the pyramid of the same side; *i.e.* he extends to these fibres the view advanced by Bouchard and by Flechsig with regard to the direct pyramidal tract.

There are on this view four ways by which the fibres of each pyramid can proceed to their ending in the cord. They proceed along (1) the lateral pyramidal tracts of the opposite side; (2) the lateral pyramidal tract of the same side; (3) the direct pyramidal tract of the same side; (4) the direct pyramidal tract of the opposite side. Most commonly all the fibres take the first course; not infrequently they run both in the first and second courses; less frequently some fibres proceed by the third, the greater number running either in the first or in the first and second course; rarely some fibres may in addition take the fourth course. If this description is the true one, the term "re-crossed" degeneration, which I have used above, is obviously inapplicable; but there are

[1] According to Sherrington a *few* scattered degenerating fibres may be present in the opposite pyramid. But I gather from his description that the number of these is wholly insufficient to account for the degeneration in the cord.

[2] This implies that the degeneration is the consequence of a previous degeneration on the opposite side of the cord—a view which though probably is not proved; the term "re-crossed pyramidal tract" used by Sherrington, is, however, still less satisfactory. A neutral term would have been best, but I could think of none.

[3] In all of these cases the degeneration had advanced to the stage of sclerosis.

reasons, which will be mentioned later, against accepting Pitres' views. Charcot's suggestion, that the fibres of one lateral tract cross to the other by the anterior commissure has, as Pitres points out, no satisfactory basis, for no degenerated fibres have as yet been described as occurring in the anterior commissure.

Bilateral degeneration in dogs after unilateral cortical injury has been described both by Löwenthal and by Sherrington.

Löwenthal mentions it as occurring in two out of about twenty cases.[1] In each of the two the degeneration (sclerosis) was diffuse, and could not be traced farther than the mid-dorsal region. Sherrington found bilateral degeneration in each of twelve cases observed three to twelve months after the lesion, but found unilateral degeneration only, in two cases observed seven and fourteen days respectively after the lesion. The re-crossed degeneration appeared to him to be always somewhat less advanced than the degeneration on the opposite side of the cord. Hence he concludes that the re-crossed degeneration begins three or more weeks after the degeneration in the opposite lateral pyramidal tract. The conclusion is somewhat hasty; for in the two cases in which re-crossed degeneration was absent, it might not have occurred had the animal been kept alive; and further, Sherrington does not appear to be quite certain that the nerve-fibres in the recrossed degeneration were really in an earlier stage of degeneration than those on the opposite side of the cord. It may be noted, however, that if his conclusion is right, it disposes at once of the views both of Charcot and of Pitres as to the origin of the fibres in the re-crossed degeneration; for if the fibres which are affected in the re-crossed degeneration were directly continuous with the pyramid fibres, they would certainly not degenerate weeks later than the rest of the pyramid fibres in the lateral pyramidal tract.

Sherrington also finds that the re-crossed degeneration does not usually diminish regularly in its course down the cord, but is greater in the upper lumbar and lower dorsal region than in the mid-dorsal region, and is commonly greater between the third to seventh cervical nerve-roots—where, indeed, it is earliest seen—than between the first to third cervical nerve-roots. If this is the case, the bilateral degeneration cannot be due to a crossed and an uncrossed portion of the pyramid, and the re-crossed degeneration cannot be due to pyramid fibres. It is on this account that I have used the term "re-crossed degeneration."

If the pyramids send no fibres to the lateral column of their own side, some other explanation of bilateral degeneration must be sought for. Let us consider in what other way it could be brought about. Since in the dog there is no evidence of a direct pyramidal tract, we have for the dog the simple scheme, that all the pyramid fibres running to the cord decussate in the pyramidal decussation, and run down the cord in the lateral pyramidal tract of the opposite

[1] It may be noticed that Homén (1883), in his account of the effects of hemisection of the cord in the dog, makes no mention of bilateral degeneration.

side. Since these fibres end in the grey substance of the cord, it is quite possible that their degeneration should cause more or less pronounced degeneration in some parts of the grey substance. And in fact one or two cases have been described of atrophy of cells in the anterior cornu in consequence of degeneration of the lateral pyramidal tract, and of atrophy of Clarke's column in consequence of degeneration of the posterior roots. And there is some evidence that in man the grey substance in lateral sclerosis is not infrequently affected in a less degree; for on the non-hemiplegic side it is not uncommon to find increase of reflexes, or general weakness without any discernible change in the columns of that side of the cord. These cases, although admitting of other explanation (see below), are most satisfactorily explained by supposing that the grey substance of the cord has been affected by the degeneration of the pyramid fibres. If the change proceeds further than this, viz. to degeneration, it is to be expected that some fibres proceeding from the grey substance to the columns of the cord, *i.e.* connecting fibres, will degenerate. Such fibres, we may fairly conclude—bearing in mind the assumption with which we started—are the fibres of the re-crossed degeneration.

But if the disappearance of the pyramid fibres on one side can affect the grey substance sufficiently to lead to the degeneration of connecting fibres on the opposite side, we should certainly expect that it would cause some degeneration of connecting fibres on its own side. There seems to me to be some evidence that this does take place. We have seen above (p. 97), that there is some reason to believe that within the area of the lateral pyramidal tract some fibres show signs of degeneration very much earlier than others. This is not easy to explain, if all the fibres are the direct continuation of the fibres of the pyramid; whereas if the fibres which are first affected are pyramid fibres, and the later ones connecting fibres of the cord, the protracted period of degeneration presents no difficulty.

Sherrington and myself have used the term "tertiary" (cf. p. 107) to denote that degeneration which is brought about by a change in grey substance as a consequence of nerve-fibres in it degenerating. We might then state the conclusions of the foregoing argument in the following way: injury to one lateral column of the cord (or unilateral injury to the motor area of the brain) causes secondary degeneration in the crossed pyramidal tract, and not infrequently tertiary degeneration of connecting fibres of the cord within the area of both crossed pyramidal tracts.

This view affords a basis for understanding how the re-crossed degeneration can, as described by Sherrington, be greater in the lower dorsal than in the mid-dorsal region; why it occurs later than the 'normal' degeneration; why on both sides of the cord fibres in an early state of degeneration are found long after the injury; and why in bilateral degeneration the area is often, as described by Pitres, greater than in unilateral degeneration. It does not satisfactorily explain how the sclerosis can be—as described

by Pitres in certain cases—equal in intensity on the two sides of the cord.

But with all this it must not be forgotten that the statements on which the view rests require confirmation; and further, that so far no relation has been observed between bilateral degeneration and atrophy of grey substance. I have stated the view with some fulness, on account of its importance, if true; but I need hardly say that I regard the questions here raised as at present quite open.

One other question of importance arises out of bilateral degeneration. This is whether each pyramid is connected with the grey substance of both sides of the spinal cord.

Flechsig, in his "Plan des menschlichen Gehirns,' figures the "direct" pyramidal tract as ending in the grey substance of the same side of the cord; those who accept Pitres' view of the meaning of bilateral degeneration might consider that here, too, each set of fibres ends in the grey substance of its own side.

That is to say, the extent of the uncrossed tracts of the pyramid would be a measure of the number of its fibres which end in the cord on the same side as it. The variability of the course of the pyramid fibres in the cord would be associated with a variability in their termination.

It is clear that there is no necessity that this should be the case. All the fibres of one pyramid might run in one lateral column, and yet end in the grey substance of both sides of the cord; and on the other hand, the fibres of one pyramid might run in both lateral and in both anterior columns, and yet end in the grey substance of one side only of the cord.

And that this is the case seems to me to be in the highest degree improbable. It requires a great deal of evidence even to make it likely that the individuals of one species vary so much, that in one the cortex of the brain is connected with one side only of the cord, and in another is connected with both sides; that, in fact, in different individuals the end stations of homologous fibres are indifferently on the right or on the left side of the body.

What, then, is the proof of this variation? It is partly that the pyramid fibres are said to vary in their course in the cord; this, even if we accept the fact, is, as I have said, no proof at all. It is partly that in some cases in which a direct pyramidal tract has been found, there has been weakness of body or increase of reflexes on the non-hemiplegic side. But this proves nothing either, for the affection of the non-hemiplegic side of the body often occurs without any abnormality being perceptible on that side of the cord.

The proof of the variation is then of the most tenuous character, and bearing in mind the objections to it on general grounds, we may safely regard it as purely hypothetical.

And we may go further, and say that in the present state of our knowledge there is little justification for believing that the pyramid fibres end on both sides of the cord. For in certain cases of hemiplegia from cortical lesion, no clinical symptoms can be

observed on the non-hemiplegic side, so that we must conclude that the pyramid fibres end on one side only of the cord. When clinical symptoms are observed on the non-hemiplegic side, they are in all probability due to an alteration of the grey substance, brought about by the degeneration of the pyramid fibres.

The Posterior Columns.—Our knowledge about the direct continuation of nerve-fibres of the posterior roots of the spinal nerves into the medulla, we owe chiefly to the observation of Bouchard[1] (1866), of Lange[2] (1872), of Schultz[3] (1883), on man, and to the experiments of Singer[4] (1881) on dogs. Schiefferdecker[5] (1876) was the first to give a detailed description of ascending degeneration after section of the cord in dogs. From these and other observations we gather the following account of the course of the nerve-fibres of the posterior root. Each posterior root on entering the cord forms the most lateral portion of the posterior column, that next the grey substance; a considerable number of its fibres at once enter the grey substance, the rest continue upwards, becoming, as they go, more mesially situated, in consequence of the entrance of other nerve-roots, and giving off, in their course, fibres to the grey substance of the cord. But not all the nerve-fibres end in the cord; some run on to end in the clava; the course of these fibres has been worked out for the sacral and lumbar nerves only. From what has been said, it is evident that the several posterior nerve-roots form ascending laminæ of fibres, so that in the upper lumbar region the nerve-root which has last entered forms a lamina in contact with the posterior cornu, and each nerve-root below this forms a lamina immediately mesial to that of the one above it, the meso-dorsal angle of the posterior column being occupied by fibres from the last sacral nerve. As these laminæ ascend in the cord, they give off at intervals, and chiefly in the dorsal region, a considerable number of their fibres to the grey substance of the cord. In the cervical region of the cord all the fibres which have reached it from the lumbar and sacral nerve-roots are found in Goll's column occupying the same relative position as they occupied lower down. These fibres, without much diminution in number, run on to the medulla, and end in the cells of the clava; they form thus an ascending medullary tract.

Hence then, broadly speaking, the nearer the mesial part of the posterior column is to the medulla, the more completely does it become an ascending medullary tract, and the less does it consist of cord fibres simply ascending from the posterior roots. It seems to me probable that it is on this account that in the developing cord—as described by Flechsig—there is in the dorsal cord no area of fibres in which the medulla is developed late, to correspond to Goll's column in the cervical cord. At the same time, one

[1] Bouchard, op. cit. t. vi. p. 463.
[2] Lange, 'Schmidt's Jahrb. d. ges. Medicin,' 1872, p. 281.
[3] Schultz, 'Arch. f. Psychiatrie,' Bd. xiv.
[4] Singer, op. cit.
[5] Schiefferdecker, op. cit. p. 589.

would expect that further observation on the time of development of the medulla of the nerve-fibres would enable the ascending medullary to be distinguished from the ascending spinal fibres in all regions of the cord. Schiefferdecker and Singer with some reservations, and Schultze unreservedly, consider Goll's column to be made up of fibres which come from the sacral, the lumbar and perhaps from the lower dorsal region. The corresponding fibres of the upper part of the cord they apparently consider run in Burdach's column. Since there are ascending medullary fibres in the lower nerve-roots, we may take it as certain that there are similar fibres and of similar function in the upper nerve-roots also. On the above view then, Goll's column contains medullary fibres from the lower part of the body, and Burdach's column contains medullary and spinal fibres from the upper part of the body. The medullary fibres of Goll's column end in the nucleus of the funiculus gracilis; the fibres of similar function in Burdach's column must then end in cells of similar function to those of the nucleus of the funiculus gracilis; thus they either leave the funiculus cuneatus for the funiculus gracilis, and end in its nucleus, or they end in the nucleus of the funiculus cuneatus; in which case the latter—in part, at any rate—has the same function as the nucleus of the funiculus gracilis, the one being the centre for the medullary fibres of the upper part of the body, the other for the medullary fibres of the lower part of the body.

Schultze's opinion is based upon an observation in man in which the middle of the cervical enlargement had been completely destroyed; the degeneration of the posterior columns, complete just above the lesion, was in no part of the cord up to the medulla confined to Goll's column, but occupied also a considerable part of Burdach's column. A similar account is given by Löwenthal of the effect of section of the posterior columns in the cervical region in the dog. These observations are not very conclusive as to the point in question; and it may be noted that no one has obtained complete degeneration of Goll's column in the upper cervical region by section of the cord in the lower dorsal region, and further, that Goll's column certainly increases in its way up the cord. We must be content to wait until observations have been made upon the effects of section of the posterior roots of the spinal nerves in the cervical region.

Since the fibres of Goll's column consist of small nerve-fibres, and since there is ground for believing that medullated nerve-fibres do not alter in size in their course in the cord, we may conclude that the medullary fibres of the posterior columns consist of a greater or less part of the small fibres of the spinal nerves, and that the spinal fibres of the posterior columns (except perhaps in the upper cervical region) include all the large fibres of the spinal nerves.[1]

[1] According to Gaskell ('Journ. of Physiol.,' vol. vi.; 1885) the *efferent small* nerve-fibres have a different function from the *efferent large* nerve-fibres. Of the function of Goll's column, nothing is known with certainty. It has been supposed to convey impulses giving rise to sense of touch (Schiff), sense of

The question of the presence of commissural fibres in the posterior columns we may omit, since the recent work on the spinal cord does not deal with it.

The Cerebellar Tract.—Singer found on section of the cord in the dog between the dorsal and lumbar regions, that in the mid-dorsal region, besides the degeneration of the tract ordinarily recognised as the cerebellar tract, there were degenerated fibres scattered over the whole of the antero-lateral column, these fibres being fewest at the periphery of the anterior column. In the cervical region the degenerated fibres were much less in number; they were present at the periphery of the lateral column only, but stretched from the anterior to the posterior nerve-roots, although they were much more diffuse ventrally of the ligamentum denticulatum than dorsally of it. These fibres in the cervical region were considered by Singer as belonging to the cerebellar tract.

According to Flechsig, the cerebellar tract in man stretches as a compact bundle of fibres about as far as the ligamentum denticulatum; at its ventral end it becomes diffuse, and scattered cerebellar fibres are found for a short distance in the antero-lateral column. Making the fullest allowance for the scattered fibres mentioned by Flechsig, there still remains a considerable difference between his description for man and that of Singer for the dog. The more recent observations on ascending degeneration, both in man and in the dog, have tended to confirm in the main Singer's account. That in man the ascending degeneration stretches more ventrally than is described by Flechsig has been pointed out by various observers, and especially by Schultze. Löwenthal has observed the ascending degeneration in the dog after section of the cord in the cervical region: according to him the cerebellar tract is fairly compact up to the denticular ligament; beyond this it becomes much more diffuse, but stretches along the periphery of the cord as far as the point of exit of the anterior nerve-roots. Near the denticular ligament it extends some little distance into the lateral column.

The fact, however, that these ventrally placed fibres degenerate in an ascending direction, is alone insufficient to show that they belong to the cerebellar tract. To show this the fibres must be traced up to the cerebellum. According to Löwenthal[1] the ventral fibres do not run in the restiform body as do the dorsal fibres, but run in the lateral region of the pons—probably as the 'aberrirende Seitenstrangbündel' of v. Monakow—twist round the superior peduncle of the cerebellum and thence run to the cerebellum.

Löwenthal, like all previous observers, finds that the degeneration of the cerebellar tract diminishes in its way up the cord;

temperature, muscular sense. Lately Herzen (Pflüger's 'Archiv,' Bd. 38, p. 93; 1885) suggests that impulses giving rise to sense of touch and sense of cold travel in the posterior columns, whilst impulses giving rise to pain and to sense of warmth travel in the grey substance of the cord.

[1] Löwenthal, 'Bull. Soc. Vaud. Sc. Nat.,' xxi. 92 (1885).

that is, that a considerable part of the 'cerebellar' tract consists of connecting fibres of the cord. Flechsig, on account of the steady increase in size of the cerebellar tract upwards, considered that it received fibres from the cord, but gave none to it; but the fact does not warrant the conclusion, for the tract must increase in size as long as it receives more fibres than it gives off. It may be mentioned that the most of the scattered degenerated fibres—probably connecting fibres—described by Singer in the dorsal region, were not observed by Löwenthal in the cervical.

According to Flechsig, Laura, Pick, and others, the cerebellar tract arises from the cells of Clarke's column. This still requires confirmation. It is somewhat against this connection that the cells of Clarke's column are of medium size, whereas the fibres of the cerebellar tract are large. An objection of a similar nature might be urged against the generally assumed connection of the pyramid fibres with chief process of the "giant cells" of the motor area of the cortex.

Fibres proper to the cord which undergo Descending Degeneration.—These fibres are called by Singer, who restricts the meaning of the term as used by Bouchard, "commissural" fibres. The term is not, I think, a very happy one. In anatomy the word "commissure" has come to mean the connecting portion between bilaterally symmetrical parts; in this sense, fibres which connect the cervical with the lumbar region of the cord do not form a commissure any more than fibres which connect the spinal cord with the cerebellum or with the cerebrum. If "commissure" is used in its wider meaning, then all these fibres are equally commissural, the fibres of the pyramidal tract, and of the cerebellar tract as much as those connecting the different segments of the spinal cord. But it would, I think, be better to restrict the term "commissural." The fibres which degenerate can conveniently be designated according to their connection and their mode of degeneration, whether above or below the point of injury. Thus we have ascending and descending spinal, medullo-spinal, ascending cerebello-spinal, descending cerebro-spinal fibres in the cord. But there are many nerve-fibres in the antero-lateral columns which do not degenerate either above or below the place of section.[1] These might conveniently be called commissural fibres. And the designation would have some justification. The fibres which degenerate have a trophic centre at one end only, and it is very likely that this is due to the nerve-fibres in life conveying impulses in one direction only, for there seems to me no reason to suppose that the fibres of the posterior roots, or the fibres of the pyramidal tracts, ever receive impulses from the spinal cord. On the other hand, the fibres which do not degenerate on section must have a trophic centre at both ends, and in view of what has just been said, it is probable that this is due to their receiving impulse at both ends.[2]

[1] Some of these may be short ascending or descending spinal fibres; the question might perhaps be determined by observing the degeneration in a piece of cord, for example, in the lower dorsal region, when completely severed from the rest.

[2] This is very nearly the view which has been put forward by Schiefferdecker, to account for the absence of degeneration in certain fibres.

If this is so, these fibres are more properly commissural than those which degenerate.

The proper cord-fibres which degenerate in an ascending direction have already been mentioned in connection with the cerebellar tract (p. 105), since the two sets have not as yet been with certainty distinguished.

Of the proper cord-fibres which degenerate in a descending direction, those which lie in, and in the neighbourhood of, the lateral pyramidal tract have also been mentioned (p. 97). The remainder are scattered throughout the ventral part of the antero-lateral column. In the dog the longer fibres form a moderately compact zone at the periphery of the anterior column. The observations of Schiefferdecker[1] and of Singer, from whom chiefly the above statements are drawn, were made after section of the cord in the lower dorsal region. In Löwenthal's experiments the spinal cord was cut through more or less completely in the cervical region; he finds throughout the lower cervical, the dorsal, and the upper lumbar region, as far as the lumbar swelling, a degenerated tract of fibres round the whole periphery of the anterior column like that previously described by Schiefferdecker and by Singer, after section of the dorsal cord. But, unlike previous observers, he finds that this tract, at the junction of the anterior with the lateral column, continues dorsally for some little distance into the lateral column, although leaving the periphery and becoming more diffuse.[2] Since these fibres diminish in number in passing down the cord, they must run to different cord segments. Since, further, the zone in the lumbar region is less compact than that previously described after section of the cord in the dorsal region, we may conclude that it receives from, as well as gives off, fibres to all parts of the cord. Some of them probably arise in the medulla, *i.e.* are descending medullo-spinal fibres.

For the fibres of short course which undergo descending degeneration, Löwenthal finds much the same distribution in the cervical region as that found by Schiefferdecker and by Singer in the lumbar region, viz. in the non peripheral parts of the anterior column, and in "anterior mixed zone" of the lateral column.[3]

Tertiary Degeneration.—Sherrington and myself found that after injury to the brain in the motor area, there was, besides the secondary degeneration in the pyramidal tracts, a degeneration of a different nature, more resembling chronic myelitis. The altered fibres were chiefly in the anterior columns, varying

[1] Schiefferdecker (op. cit. p. 569) considered these fibres to correspond with Türck's 'Hulsenvorderstrangbahn,' in man. They have, however, nothing to do with the pyramids.

[2] These, together with the corresponding fibre of the anterior column, Löwenthal calls "long commissural fibres." If this term is used, it must not be confused with Bouchard's anterior long commissural fibres, which lie in the dorsal part of the lateral column (cf. p. 97).

[3] Schultz (op. cit.) describes in man, after a lesion in the cervical region, degenerated fibres throughout the anterior column and anterior mixed zone; in the mid-dorsal region the degenerated fibres were confined to the inner half of the anterior column.

somewhat in position in different parts of the cord. Since this was apparently the result of an alteration in the grey substance of the cord in consequence of the secondary degeneration of the pyramidal tracts, we called it a tertiary degeneration, at the same time pointing out the possibility of its having been produced by the hardening agent. Sherrington has since found two other cases in which, after cortical lesion, a similar change had taken place in the spinal cord. As he points out, the entire absence of altered fibres from the cerebellar tract is strong reason for regarding the "myelitic" appearance of certain fibres in the rest of the cord as being due to degeneration, and not to the hardening agent.

The Parts of the Cortex of the Brain which are connected with the Lateral Pyramidal Tracts.—It is well known that a lesion which affects a considerable part of the motor area of the cortex of the brain causes a degeneration in the lateral pyramidal tract of the cord, and that a considerable lesion in other parts of the cortex—such as that of the occipital and temporal lobes—causes no degeneration in the cord.

We know very little more than this, but there has been a great tendency to consider this knowledge as much more precise than it really is. This is so because it is commonly assumed: 1st. That the motor area of the cortex, as it has been determined by electrical stimulation, coincides with the "cord area," *i.e.* with the area of the cortex, destruction of which causes degeneration in the cord. 2nd. That each "centre" in the motor area is connected with its corresponding local centre in the spinal cord, and with that only; for instance, that the cortical area, stimulation of which causes movements of the fore-arm, is connected with the "arm centre" in the cervical region of the cord, and with no other part of the cord.

Both of these assumptions follow so naturally from the results of cortical stimulation that it may cause some surprise that I should touch upon them. But it is necessary to do so in order to discuss fairly the results of cortical extirpation.

Ferrier and Yeo,[1] in a very interesting paper on the physiological effects of extirpation of the cortex in the ape, touch somewhat incidentally on the question of secondary degeneration. In the cases in which the cord was examined, secondary degeneration was only found when the cortical lesion had taken place in the motor area (the convolutions bounding the fissure of Rolando). These observations are distinctly in favour of the view, that the "cord area" coincides with the motor area for the limbs and trunk, but the details given are not sufficiently full to prove that this is the case.

The more recent experiments in the dog are difficult to harmonise with the scheme given above.

Sherrington and myself, from a comparison of the area of greatest degeneration on the two sides of the cord consequent on

[1] Ferrier and Yeo, 'Phil. Trans. Roy. Soc.,' pt. ii., 1884, p. 479.

destruction of the cortex of the brain to unequal extents and at different times, were inclined to believe that the anterior part of the motor area was connected with the dorsal part of the lateral pyramidal tract, and the posterior part of the motor area with the anterior part of the lateral pyramidal tract; and further, that the destruction of the cortex behind the motor area as figured by Hitzig and by Ferrier would give rise to degeneration in the cord. The nature of our observations did not, however, allow of conclusions being drawn with certainty. It will be noticed that our tentative conclusions involved: (1) That fibres from each "centre" of the motor area run in a fairly compact bundle. (2) That the cord received nerve-fibres from parts of the cortex which are considered to be motor centres for certain of the muscles of the head. (3) That the "cord area" stretches farther posteriorly than the described motor area.

The posterior part of the cord area—as given above—lies in part, at any rate, in the region which, in Ferrier's observations, produced, on stimulation, movements of the eye. Ferrier considered it, however, to be a part of the "sensory" area of the cortex. This region is called the "Fühlsphäre" for the eye by Munk, who puts it in the same class with the cortex of the sigmoid gyrus, which he calls the "Fühlsphäre" for the limbs and neck.

Löwenthal's observations deal with these questions. He has examined the brain and cord of a considerable number of dogs from which various parts of the cortex had been removed by Schiff. Unfortunately the lesions were in too many instances insufficiently localised.

Löwenthal has observed five cases in which the coronal and anterior ecto-sylvian convolutions—the anterior parts of the 3rd and 2nd convolutions—were largely removed; in two of these cases a part also of the median ecto-sylvian and of the anterior sylvian convolution were also removed. In each case, however, a small part of the postero-lateral edge of the sigmoid gyrus was affected; in one case this appears to have been a mere fraying of the edge of the sigmoid gyrus. In every instance there was a degeneration of the lateral pyramidal tract of the spinal cord, a degeneration which could be followed a variable distance in the dorsal region.

If the degeneration was due to the destruction of the coronal, ecto-sylvian or sylvian convolution, it follows that the destruction of the motor centres for certain muscles of the head, causes degeneration of part of the lateral pyramidal tract of the cord. It is unlikely that the slight and superficial injury to the sigmoid gyrus should have caused so considerable a degeneration as occurred in these cases; but even by granting this, the difficulty is not removed, for the part of the sigmoid gyrus injured[1] is the motor area for the fore-limb, and the destruction of this ought not, on the generally accepted view, to cause degeneration in the dorsal region of the cord.

[1] Paneth, in his recent paper, places one of his small motor areas for the hind-limb near this spot.

It may be, however, that in each of these cases the lesion was carried too deep, and affected part of the corona radiata containing fibres coming from the cortical "centre" for the hind-limb. Löwenthal mentions this possibility, but does not say that there was any evidence for it in the sections of the brain.

Sherrington gives an observation which is more difficult to explain on the ordinary view of the connection of the motor centres with the cord. He finds that a lesion which does not reach farther forward than the posterior edge of the ecto-sylvian fissure, produces degeneration throughout the cord, *i.e.* a lesion which lies posteriorly to the motor area as given by Hitzig, Fritsch, and by Ferrier,[1]—though including part of the "Fühlsphäre" of the eye of Munk—causes degeneration in the lateral pyramidal tract. In his case it is very difficult to see how fibres from the sigmoid gyrus could have been affected.

Further, Löwenthal finds that lesion of the anterior limb of the sigmoid gyrus—the posterior lateral border being left intact—leads to degeneration as far as the upper dorsal region.[2] No one, I believe, has considered the anterior limb of the sigmoid gyrus to contain a motor area for any of the muscles of the trunk or of the hind-leg.

From the destruction of certain parts of the cortex, Löwenthal has observed no degeneration in the cord. In a case in which the lesion was confined to the postero-dorsal part of the sub-orbital lobe degeneration was absent. Sherrington also found no degeneration from injury of the sub-orbital lobe. According to Munk, the sub-orbital lobe is the "Fühlsphäre" of the trunk.[3]

In another case described by Löwenthal, in which degeneration was absent, a roundish area of the cortex was destroyed, involving the mid-third about of the ectolateral convolution, by passing a short distance over the lateral fissure into the fourth convolution, and nearly reaching the supra-sylvian fissure. In a third case the roundish area destroyed was a little larger than the preceding; cutting across the median ectolateral (2nd) convolution, it just passed over the supra-sylvian fissure dorsally, and nearly reached the sylvian fissure ventrally; a prolongation of the sylvian fissure would have nearly bisected the area of the lesion.

The tentative conclusion mentioned above, that the fibres from different parts of the motor area run in different parts of the lateral pyramidal tract, has received no confirmation. Löwenthal examined the cord in seventeen cases of cortical lesion affecting the sigmoid gyrus in different degrees and in different regions. He mentions no difference in the cord degeneration, except a difference in intensity. Sherrington finds the position of the cord degeneration to be the same whatever the cortical lesion which

[1] Luciani and Tamburini also place the motor areas for the trunk and limbs in the sigmoid gyrus.

[2] The lower dorsal and lumbar regions were not observed.

[3] Ferrier ('Transact. Royal Soc.' 1884, p. 528) mentions that in the ape, destruction of the frontal lobes anterior to the motor region, causes degeneration of the mesial part of the crusta, but none in the pyramid.

has given rise to it. If this is so, it shows a very remarkable intermingling of the fibres of the pyramidal tract in their course from the cortex.

A limited intermingling of fibres is suggested by the observations of Hitzig, who found that movements of the muscles of the trunk could be obtained by stimulation of the motor area for the fore-limbs; and a greater intermingling is suggested by the recent observations of Paneth[1] made under Exner's direction. Paneth finds that there are small motor areas for muscles of the fore- and of the hind-limbs interspersed over a considerable area of the posterior limb of the sigmoid gyrus.

But both as regards this point, and as regards the connection of the part of the motor area with the lateral pyramidal tract, we must wait for further investigations. The experiments which have been mentioned above suggest conclusions which are not in accordance with those which we should on general grounds be inclined to accept; for this reason, then, we are justified in reserving a definite opinion until fuller and more satisfactory evidence is forthcoming.

[1] Paneth, 'Pflüger's Arch.,' Bd. 37, p. 521 (Dec. 8, 1885).—See Abstract in the present number of 'BRAIN,' p. 144.

ON SOME RECENT ADVANCES IN OUR KNOWLEDGE OF THOMSEN'S DISEASE.

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(1.) 'A Consideration of the Thomsen Symptom complex, with reference to a new form of Paralysis Agitans.' By ALLAN McLANE HAMILTON, M.D. *The Medical Record*, New York, January 23, 1886.

(2.) 'Klinisches und Pathologisch-Anatomisches von der Thomsen'schen Krankheit.' Von Prof. W. ERB. *Neurologisches Centralblatt*, No. 13, 1885.

(3.) 'Thomsen'schen Krankheit bei vier Geschwistern.' Von Prof. EULENBURG und Dr. MELCHERT. *Berliner Klinische Wochenschrift*, No. 38, 1885.

(4.) 'Une Observation de Maladie de Thomsen.' Par MM. PITRES et DALLEDET. *Archives de Neurologie*, No. 29, 1885.

(5.) 'Ein Fall von Thomsen'schen Krankheit.' Von DR. G. FISCHER. *Neurologisches Centralblatt*, 15th February, 1886.

(6.) 'Maladie de Thomsen et Paralysie pseudohypotrophique.' Par VIGOUROUX. *Arch. de Neurologie*, vol. 8, No. 24, Nov. 1884.

(7.) 'Observation d'un cas de Maladie de Thomsen.' Par L. DELIGNY. *L'Union Médicale*, 1885, No. 5.

(8.) 'La Maladie de Thomsen (Dysmyotonie Congénitale).' Par LONGUET. *L'Union Médicale*, 1883, November 25.

(9.) 'Contribution à l'histoire de la Maladie de Thomsen.' Par P. MARIE. *Revue de Médecine*, 1883, p. 1064.

(10.) 'Fem Tilfälde af den Thomsen'ske Sygdom. Af KNUD PONTOPPIDAN' (*Hosp.-Tidende*, 1884, 3 R. II., 34).

(11.) 'Ueber eine dem Bilde der cerebrospinalen grauen Degeneration ähnliche Erkrankung des centralen Nervensystems ohne anatomischen Befund, nebst einigen Bemerkungen über Paradoxe Contraction.' WESTPHAL, *Archiv für Psych.*, Bd. XIV., p. 87 (especially p. 130).

(12.) 'Malattia di Thomsen. Sua Analogia coi fenomeni muscolari dell' ipnotismo,' Sepilli, *Arch. Ital. per le malattie nervose ec.* Fasc. v. 1885.

(13.) 'La Malattia di Thomsen.' PETRONE, *Lo Sperimentale.* July, 1884.

(14.) 'Handbuch der Kinderkrankheiten.' GERHARDT, t. v., p. 175.

(15.) 'A Case of Thomsen's Disease; a form of Paresis of Motion, accompanied by muscular hypertrophy.' ENGEL, *Philadelphia Medical Times*, September 3, 1883.

(16.) 'Thomsen's Disease.' By PAUL CHAPMAN, M.D. *Brain*, vol. vi.

In the article last mentioned there is an almost complete list of the literature of the subject up to that date, and I think the fifteen other references that I have enumerated made the whole of the literature very nearly, if not quite complete up to the present date. The first five of the above articles will be discussed in detail, for not only are they the most recent, but also the most important. The sixth article does not call for comment, and the seventh relates an example of the disease in which the patient was a man 25 years old; his father had had the disease, but his general health had in no way been affected by it. The patient was the only child; the cramps caused great pain, and came on several times a day, and lasted a long while; the reflexes were normal. The peculiarity of the case seems to be that the contractions lasted so long, and there was so much pain with them.

The eighth article gives a very good account of the literature of the subject up to date, but makes confusion worse confounded by using another term, viz. dysmyotonie congénitale, almost adopting Strumpell's term, congenital myotonia. The exact nature of the case in the ninth article is probably rather doubtful, for the cramps were strictly one-sided, and the patient also suffered from epileptiform seizures. The tenth article records five cases, two in one family, and three in another. Portions of muscle were examined microscopically, and are stated to have been healthy. This paper is to be found abstracted in the 'Neurologisches Centralblatt,' 1884, p. 520.

In the eleventh of the above-mentioned papers Westphal discusses briefly the nature of paradoxical contraction, and comes to the conclusion, that the contraction of Thomsen's disease is allied to it, but that our knowledge about both of them is at present very elementary.

Petrone's account of the disease is good, brief, and to the point, but does not contain anything new about it.

The first of the above articles begins with a historical account of the malady. It is pointed out, as is well known, that Sir Charles Bell, in 1836, mentioned the existence of a disease which was probably the same as the one now under discussion, but gave it no distinct name. The next person to call attention to it was Thomsen, who in 1876 described the disease as occurring in members of his own family. Since that time many examples of the disease have been recorded, chiefly in Germany, together with a few in each of France, Sweden, and Italy, and two in America. Altogether about twenty to thirty cases have been recorded. Hamilton gives the clinical expressions of the disorder as follow:—(1) a sudden spasmodic muscular seizure which attends the performance of a voluntary act; (2) its subsidence after the repetition of the act; (3) an apparent increase in bulk in several muscles which are chiefly and most frequently the subjects of cramp; (4) an increase in electro-muscular irritability; (5) a normal or slightly excited state of tendinous reflex activity; (6) these symptoms are often of congenital origin, and progressive as regards intensity.

I shall show later on that all these symptoms are in the main

correct, but that by the light of our present knowledge they can be much more definitely described. Hamilton points out that the lower extremities are most often implicated, and that hence when the patient essays to walk, there is sudden flexion of the thigh and leg, in which position, owing to the stiffness in contraction of the muscles, they remain in this condition from five to ten seconds, so that the patient often falls. It may be some time after a volitional impulse that the muscle contracts, when it does there is this stiffness [Muskelssteifigkeit]. The muscles of the face may be affected. Cold and mental excitement increase the liability to the disease. In only one case were the reflexes exaggerated; a congenital history is not always the rule. Hamilton's object in writing the paper is to throw doubt upon the question whether Thomsen's disease is to be looked upon as a distinct malady, for he points out that this muscle-stiffness on contraction is a frequent symptom in a variety of maladies; in most cases it is purely psychic; it is an occasional symptom of the third stage of locomotor ataxy; it is common in hystero-organic disorders of the nervous system, and may be associated with coarse cerebral lesions, or may be present in Parkinson's disease. No mention is made of the recent article by Erb and others, or of the microscopical appearance of the muscles, nor are the electrical reactions and mechanical irritability reactions referred to in much detail; and although the question is still under discussion, and awaiting a promised article by Erb on the subject, I think we shall see that whilst it is by no means deniable that cramps may take place in a number of diseases, still this group of symptoms taken with the changes in the muscle, points strongly to the fact, that Thomsen's disease must be looked upon as a genuine and distinct malady. In further proof of this it may be observed that the hitherto recorded cases have not been suffering from paralysis agitans, nor any other disease mentioned by Hamilton. The author then gives an account of a boy, aged 10 years, who had always suffered from cramps, which were worse under mental excitement, and caused him to walk in a squat position. No change could be detected in the portions of muscle removed, and the electrical reactions were normal. Both these facts are against the case being one of Thomsen's disease, and no diagnosis seems to have been made. Another case is given in which a boy, 9 years old, who had suffered from headache and attacks of vomiting, had muscle-stiffness on contraction in the voluntary muscles; muscular enlargement and loss of power; the mouth was drawn slightly to the left side; the tongue pointed to the right; there was a prominence of the eyes and a slight lateral nystagmus. The headache and vomiting do not necessarily preclude Thomsen's disease, as the cases of Eulenburg and Melchert show; still the other symptoms probably do, and so we have here another example of cramps occurring in other diseases. For the convenience of those who wish to look up the subject, I may mention that Thomsen's original article is to be found in the 'Archiv für Psychiatrie und Nervenkrankheiten,' 1876. Hamilton, at the heading of his article, uses the phrase "Thomsen symptom complex." This is a pity, for not only does it lead to con-

fusion, because the phrase "symptom complex" has a wider significance, as we see in an article by Westphal, "Ueber einen eigenthumlichen Symptomencomplex bei Erkrankung der Hinterstränge des Rückenmarks,"[1] where the "Symptomencomplexes" apparently included paralysis, paradoxical contraction, rigidity and cramp in certain movements; but a much better phrase, viz., myotonic contraction, has been proposed, which is shorter and less cumbrous than "Thomsen symptom complex."

Erb begins his article by pointing out, that although there has never been a post-mortem examination on a case of Thomsen's disease, yet the five observers who have excised portions of the muscle have failed hitherto to find any change in it. He gives an account of two brothers, aged respectively 14 and 15 years, who had it typically, and in whom it had been present some time. The voluntary power of the muscles was slightly diminished, especially considering their size. On movement of the voluntary muscles there was the usual trouble, viz. a slowness and difficulty of contraction; then a stiffness and tension, which suddenly passed off; and then there was free movement. With a very powerful voluntary movement the duration of this tonus was from 10–25 seconds. The motor nerves with mechanical stimulation showed diminished excitability. With the faradic and galvanic currents the reactions of the nerves are practically normal, unless the stimulations follow very quickly, when there is a slight lasting contraction. In the muscles the mechanical excitability is increased, and after a strong stimulus remains from 12 to 30 seconds. A strong faradic current gives a contraction which remains for 20 or more seconds; but on opening, there is only a momentary contraction with even the strongest current. With the galvanic current the muscles usually give only closing contractions. K and A are usually alike, but in some muscles Ka S Z may be best, in others An S Z. It is with Ka S Z that one gets the best tonic contraction, which is often extremely good, and lasts 5–30 seconds. With the stimulations rhythmically following one another, we get wave like contractions also following each other, and going along the muscle from the kathode to the anode. This is well seen in the flexors of the fingers, the vastus internus and externus. All the above reactions are best shown in muscles which are under the influence of the will. The essentials are the slowness and duration of the contraction; a good name for it is myotonic contraction or myotonic disturbance of muscles. It shows itself on direct stimulation of the muscle, or, in the case of the nerve, when one stimulus quickly follows another.

On microscopical examination of a piece of excised muscle in the fresh state, the only thing that could be made out was that the fibres were a little broader than natural; but with Müller's fluid preparations they appeared many times wider than they should be, and not polygonal, but rounded. The interstitial tissue and vessels were normal; there was considerable thickening of the sarcolemma.

[1] 'Archiv für Psych.,' etc., 1885, Bd. xvi.

Eulenburg and Melchert have given a most interesting account in which the disease affected four brothers and sisters. The family history was unimportant; the mother suffered from cramp in the stomach and headache. Of the six children, all suffered from cramps and convulsions during teething, and during their school-days from hemicrania and vomiting. One daughter had migraine; another used to wake up with attacks of cardiac pain and palpitation. The three sons and the eldest daughter were typical examples of Thomsen's disease. In all of them the voluntary muscles were affected, and there was the usual contraction. The following are the details of each of the four cases.

W. K., 22 years old; he was first attacked when 7 years old, and then he found stiffness on attempting to run; this continued off and on, and from 14 to 18 he suffered very badly, so that he would often fall in the street; after 18 he became a little better; but during his service in the army the disease gave rise to so much trouble, that he had great difficulty in carrying out correctly the exercise orders; in the same way it was difficult, in shooting, to raise the musket to the shoulder, and he had much difficulty during his apprenticeship as a dentist. On examination, the peculiarities of contraction of voluntary muscles, as described in the two previous articles, were most apparent, and, as already mentioned, after a few contractions the myotatic contractions passed off. The peculiarity was present in the forearms, lowers, body, face, deglutition muscles, tongue and eye muscles. The myotatic contraction was most marked the longer the muscles had been at rest, worse after cold and inanition, but better after meals. On direct mechanical stimulation, after a short time the myotatic contraction appeared, lasting 3–5 seconds. With faradic stimulus to muscle and nerve there was nothing noteworthy. With the galvanic current to the muscles, K S Z appeared sooner than A S Z and A O Z > K O Z, but the contractions came slowly, and lasted 2–5 seconds; with galvanic current to the nerve, K S Z appeared sooner than A S Z, but the contraction was momentary. Patellar reflex weak.

George K., 21 years old; as a child had headache and vomiting. When a boy, had slight difficulty of movement, which showed itself in gymnastics. His muscles were, when he performed voluntary acts, in the same myotatic condition as those of his brother, but worse. Mechanical stimulation to muscles gave the same results, but they were more pronounced. Faradism as in brother; galvanism to nerve K S Z > A S Z, with no myotonic contraction; this and muscular galvanism also being the same as the brother. There was a prolonged latent period; there was in succession a prolonged contraction, and then a sudden relaxation, best seen after prolonged rest; frequent slight tremors of the muscles were to be seen, and at times there were very strong opening contractions with the kathode.

Carl K., 21 years old. Headache and vomiting when young. The trouble with the muscles came on at 12 years, when he was found to be bad at gymnastics. The symptoms were not so

marked in his case; the mechanical myotatic contraction was present in slight degree. Faradism gave no lasting contractions; the galvanism results were just the same, but weaker. Patellar reflex weak.

Louise A., 29 years old. The disease first appeared in childhood, and got worse at puberty. She has great difficulty in going upstairs, and in executing complicated movements, as dancing. All the symptoms were pretty much the same as in the brothers. The author directs attention to the fact, that the two children who have not had Thomsen's disease have anomalies of their nervous system; also that the symptoms increase after cold and exhaustion, and the daughter was better when she lived in more comfortable circumstances. The four cases showed slight qualitative differences to their electrical reactions, and Erb's wave contractions with strong current were not observed, but there was a tendency to tremulous oscillations with faradic stimulation.

MM. A. Pitres et Dalledet have given a very full account of a patient suffering from this disease; they also give several myographic tracings. The grandparents and the father were healthy. The mother at the age of 15 suffered from muscular stiffness, like that which her son, the patient, suffers from now. This difficulty disappeared in two or three years; then there were cramps, chiefly in the sacro-lumbar region, the pains coming on day or night, without any relation to movement. She and her husband had six children; the three first being like the father in all points, the three younger, on the contrary, suffering from muscular stiffness on voluntary movement. Of these, the fifth is the patient, and will be described in detail; the fourth, a man of 26 years, is exactly like the patient in all the symptoms, but not so bad; and the sixth, a girl of 19 years, had a difficulty of voluntary movements when 15 and 16, but that has now disappeared. The difficulty of voluntary movement of which the patient complains began when he was a child. Up to the age of 22 it increased but slowly; since then it has gone on very quickly. He is now 24.

The first thing that strikes one is the hypertrophy of the muscles, which is well seen in the arm and forearm, and the thenar and hyperthenar eminences. The sterno-mastoids, triceps of thigh, and sacro-lumbar muscles are all hypertrophied. During contraction they are irregularly bossed, and as hard as wood. The power of the muscles is not so great as their hypertrophy would lead one to expect. The electrical reactions are normal. The difficulty of voluntary movement consisted in a restraint and stiffness on the attempt, which gradually passed off as the movement was frequently repeated. This is very well shown by a myographic tracing, which shows that the latent period is increased considerably, and the muscle remains contracted much longer than it should. The authors also insist on a point which their tracings show, but which has not been noted in other cases, namely, that not only is there a difficulty of contraction, but also of the first *décontractions*; in fact, in the tracings the down-stroke

often extends over a longer period than either the up-stroke or the crest. With a strong and rapidly repeated faradic current, the same thing is observed in the early contractions. This, it will be observed, brings this case somewhat in harmony with Erb's electrical myotatic contractions, and perhaps when the authors here speak of the electrical reactions normal, they did not look for the myotatic reactions. The peculiarities of contraction were increased by cold. The muscles of the face would sometimes remain in an emotional expression so long, that the hand had to be used to aid their return to normal, hence, sometimes there was difficulty of speech. The fear of the peculiarity being observed increased it. The tongue, deglutition, phonation, respiration and ocular muscles were not affected. Passive movements were easy, and sensibility was intact; reflexes normal, sensation of position in space normal. All other functions of body healthy.

The fifth paper, mentioned at the beginning of this notice, has only just been published. The author observes that in this case the skin and tendon reflexes were normal, and that the strength of the muscles was not so much increased as would have been expected from the increase of volume. Sometimes the myotonic contraction remained from 25–27 seconds after voluntary contraction, but after repeated movements it disappeared. On the whole, this case agrees with others, and hardly differs more from them than they do among themselves. The myotatic reaction, in response to direct muscle stimulation, was not so easily obtained, and no K O Z could be obtained on stimulation of the nerve. The rhythmic wave-contractions of Erb were shown very well indeed, and with a current of a strength of 15–20 M A, they were well formed, slow, and about 15–20 seconds apart, passing from the kathode and the anode. With a strong faradic current there were rapid oscillations of the muscles. On microscopic examination of the fibres they are found to be thickened. In no parts was the cross striping well marked. The contour of the bundles is uneven, and has here and there little tumefaction. The author raises the question whether these appearances are due to the mechanical stimulation of excision.

In conclusion, we may, I think, gather that the following are the chief characteristics of Thomsen's disease:

1. It occurs in young subjects, coming on usually in youth or childhood.
2. It lasts a long while; is never fatal; and may pass off.
3. The muscular difficulty is much increased by cold and mental excitement.
4. It occurs in members of the same family, and is slightly hereditary.
5. Other neurotic disturbances in the family are by no means always well marked.
6. Voluntary muscles only are affected, the legs perhaps oftenest.
7. There is an increase of bulk in the muscles.

8. But considering their increase in size, they are not so powerful as might be expected.

9. On attempted voluntary movement the latent period is usually prolonged; then comes a severe contraction, lasting from five to thirty seconds; then in some cases a sudden, and in others a slow relaxation. This peculiarity constitutes a myotonic contraction.

10. The above is more marked, the longer has been the interval of rest. After repeated contractions the difficulty becomes less, and soon passes off, the contractions being ordinary.

11. Mechanical stimulation of nerves shows a diminished excitability. Faradic or galvanic stimulation of nerves gives slight myotonic contractions on being rapidly repeated.

12. Mechanical stimulation of muscles shows increased excitability, with long myotonic contractions. Galvanic stimulation usually gives closing contractions; K S Z is generally the best, giving a good myotonic contraction, but the exact order is variable. Strong faradic current on closing gives a good myotonic contraction; on opening, a very slight contraction.

13. With stimulations rhythmically following each other, we get wave-like contractions passing along the muscle from the kathode to the anode.

14. The reflexes are normal.

15. There is increased width of the muscle fibres, which lose some of their striation, are rounded instead of polygonal, and have protuberances on their contour. The sarcolemma is thick.

Reviews and Notices of Books.

I. *Beiträge zur Anatomie und Physiologie.* Von Prof. C. Eckhard (Giessen). Vol. X. No. IV. *Geschichte der Experimental Physiologie des Froschhirnes.*—4to. Giessen: Emil Roth, 1883.

II. *Untersuchungen über die Physiologie des Froschhirnes.* Von Dr. J. Steiner (Heidelberg). 1 vol. 8vo. pp. 143. Braunschweig: Vieweg, 1885.

III. *Saggio Sperimentale sul meccanismo dei movimenti voluntari nella Testuggine palustre.* Del Prof. Giuolio Fano. 1 vol. large 8vo., pp. 61. Firenze: Lemonnier, 1884. *Sul nodo Deambulatorio Bulbare.* Del Prof. G. Fano. 1 vol. pp. 23. Genova, 1885.

In the second half of the 17th century, Robert Boyle in England, and Francesco Redi in Italy, performed the first experiments on record, with a view to determine the functions of the brain in amphibians and reptiles. In his essay on "The Usefulness of Experimental Natural Philosophy," published in 1663, the former appears to be conscious of the fact, that the ablation of the frog's brain abolishes spontaneous movements, whilst the latter observed a similar phenomenon, though not so constantly, after excerebration of tortoises. Other early observers repeated the experiment without adding materially to these results. Humboldt (1797) appears to have been the first in the application of galvanic stimuli to the cerebral centres, but without obtaining any motor effects.

Rolando (1809) found that integrity of the medulla was a necessary condition for the keeping alive of cold-blooded animals after complete excerebration; and that after removal of the hemispheres only, animals moved fairly well on external excitation. The cerebellum he thought to be an organ of locomotion, comparing its lamellated structure to the recently discovered voltaic pile. Three years later Legallois published his 'Expériences sur le principe de la vie,' in which we find some criticisms upon the imperfect methods followed by his predecessors. He introduced the method of successive sections across the encephalon, and so was able to determine the seat of the respiratory centre in the medulla. Legallois made numerous experiments on the cerebral innervation of the heart, a subject still more fully worked out in Wilson Philipp's papers, published in the 'Philosophical Transactions' for 1815.

It was Flourens who first attempted to define the functions of

the several divisions of the encephalon. The first edition of the 'Recherches expérimentales sur les propriétés et fonctions du système nerveux dans les animaux vertébrés" appeared in 1824. He found that the ablation of both hemispheres was necessary to deprive the frog of its motor spontaneity and its sensibility; but afterwards he qualified the latter statement by distinguishing between "sensation" and "the perception of sensations, or intelligence." He maintained that rotatory movements follow removals of the optic thalamus and optic lobe; in the first case they are towards the side of the lesion, in the second in the opposite direction. His well-known discovery of the coördinative function of the cerebellum was not made on the frog, in which this organ is rudimentary. Coster, a pupil of Rolando whose name Flourens does not mention, made a violent attack upon the latter's originality. Though Flourens never denied having read the works of his predecessor, Magendie had no difficulty in showing the wide difference existing between the methods and results of the two observers.

Magendie, Desmoulins, and Serres, confirmed, whilst they partially corrected and extended, the conclusions reached by Flourens. Paton, in a paper published in the 'Edinburgh Medical Journal' (1846), described for the first time the phenomenon since known as the Goltz's "Quackversuch," and which consists in eliciting a croak from a frog deprived of its hemispheres, by stroking it over the interscapular region. Other observers, such as Cayrade (1864), studied the swimming movements of the similarly prepared animal, and the question whether it could "see" gave rise to many discussions, in which, among others, Renzi (1863), Goltz (1869), Vulpian (1866), and Blaschko (1880), took part. It was shown that the hemispheres are not necessary to enable the frog to avoid, or jump over, obstacles; but how far these actions are conscious, and not merely coördinated and adapted visual reflexes, is a problem to be settled only in the light of more extensive researches in comparative psychology. The same remark applies to other reflex phenomena, where the excitation is conveyed through the auditory or tactile organs, and which are found not to be abolished by the ablation of the hemispheres.

Dr. Steiner's book constitutes the most recent contribution to the subject now before us, and is a model of what a work of its kind should be. In the introductory chapter we find a clear statement of the conditions under which experiments on the frog's brain are to be conducted. The author insists upon the necessity of giving the animal perfect rest after the operation, and of then examining its behaviour both on land and in water. He speaks of the importance of accuracy in producing the lesions, and in defining them post-mortem, and accepts Goltz's expression of "Ausfallserscheinung" to denote the symptoms due to the absence of a portion of the brain, in opposition to that of "Nebenwirkung," or "Reizungserscheinung," which is applied to the effects of the traumatism—terms which could be rendered in English by

"direct," or "essential," and "indirect," or "concomitant," respectively—a distinction equivalent to that made between destructive and irritative lesions in human pathology.

Dr. Steiner has studied carefully the conditions under which the frog, deprived of its *hemispheres*, can be made to jump over an obstacle, instead of avoiding it, and proves the importance of visual impressions in the execution of these acts. Goltz's "Balancirversuch" is fully discussed. When the animal is placed upon a small board, which is gradually tilted upwards, it begins to ascend, and finally rests upon the edge. On continuing the movement, and so lowering the board in the opposite direction, the frog begins to descend, raising its head, instead of depressing it, as during the ascent. These phenomena are purely reflex, and depend, as Dr. Steiner shows, upon different impressions, due to the strain upon the muscles and joints of the head and neck, arising from altered equilibrational conditions.

Careful observation of the behaviour of the prepared frog in water, proves that the influence of vision has hitherto been insufficiently taken into account. Placed in a quadrangular basin with perpendicular walls, the animal makes for one of the corners; then directing its eyes upwards, takes a spring that lands it on the edge, where it assumes the characteristic motionless attitude. Here, as in the experiments with obstacles, conditions of light and height are necessary to obtain these results; the frog does not attempt to jump unless the light shines from above, and the top is not too high to be reached.

The effects of a removal of the *thalami* (in addition to that of the hemispheres) does not seem to have been thoroughly investigated before Steiner. Within two or three hours the skin, even when of the lightest yellow, turns to a dark-brown tinge, which persists till the death of the animal. The movements, both on land and in water, which ensue from peripheral excitations, are somewhat clumsier, and there is no ascent of the inclined plane, the head alone executing the customary motion. The frog, however, still turns over when placed on its back. Unless the optic nerves have been divided at the same time, removal of the thalami does not appear to exert any influence upon the visual reflexes, and the organs appear to be connected with musculo-articular, rather than with cutaneous feelings.

The *optic lobes* of the frog are the homologues of the mid-brain of the higher vertebrates, and consist of two rounded masses containing a ventricular cavity. We must distinguish between their upper and lower parts, which we may designate as "roof" and "base" respectively. The roof represents the corpora quadrigemina in man, and Renzi (1863) has shown it to be necessary to vision. Cayrade's experiments tended to show that the optic lobes of the frog have functions similar to those of the cerebellum in the higher animals; while those of Goltz demonstrated that whereas certain reflexes (Quack- and Balancir-versuch) disappear with the ablation of these organs, others (turning-over, reaction to cutaneous excitations) still persist. Newell Martin (1878) states

that under the same circumstances the respiratory movements are diminished, but become tetanic on excitation.

Dr. Steiner shows that the utmost care is required to obtain trustworthy results from the ablation of the optic lobes. A frog so prepared still jumps fairly when touched, but has lost some of its swimming powers. In their normal condition frogs swim in two different ways; in the first, or fully coördinated mode, they hold the fore-legs adducted and drawn backwards along the side of the body, whilst the hind-legs perform energetic movements of flexion and extension; in the second, the fore-legs are kept semi-flexed in front, the hind-legs being used in a more leisurely, paddling way. Now ablation of the lobes reduces the swimming powers of the animal to the second mode.

When the anterior two-thirds only of the optic lobes are removed, a curious phenomenon is observed; on exciting the frog in the usual way (*i.e.* pinching the toes of the hind-leg), it begins to walk backwards. Dr. Steiner very justly remarks that this does not imply the existence of a special centre for that movement, but merely a disturbance of the afferent part of the mechanism upon which the coördinated reflex depends. The same remark applies to the altered mode of natation, which is observed likewise on partial ablation of the lobes.

It is very probable that the effects ascribed by previous observers to a removal of the frog's *cerebellum*, were due to inaccuracies in the performance of the operation. When the latter is strictly limited to the part, none but slight disturbances of movement ensue. The animal performs the usual ascent of the slanting board, but on its making efforts, a certain unsteadiness and tremor is noticeable. When it jumps out of the water upon the edge of the basin, the spring is not accurately measured, being either too long or too short. Removal of the cerebellum from an animal already deprived of its optic lobes, gives rise to no additional symptoms, a fact not surprising when we consider the small size of the organ.

It thus appears that a frog deprived of every part of its encephalon except the medulla oblongata, is able to preserve its natural posture when at rest, to jump, to swim, to right itself when placed on the back. It is true that earlier observers (Rolando, Renzi, &c.) went further, and thought the animal was still conscious of sensations; but we know how fallacious are all such conclusions drawn in the lower animals from apparently purposive reflex movements. Now the localisation of those motor mechanisms is in the anterior part (pars commissuralis) of the bulb which lies under the lamella-like cerebellum; for when a section is made at the level with the posterior edge of the cerebellum, the frog remains limp, whether on land or in water, and no longer reacts to excitations by coördinated locomotor movements. When placed on a rotating disk, it no longer turns its head to one side, as is otherwise the case, nor does it turn round when placed in the dorsal position. The commissural portion of the bulb must contain motor centres for the muscles of the head,

limbs, and trunk, which are brought into play by peripheral excitations and by disturbance of the equilibrium of position. Dr. Steiner further shows that these centres are in direct organic relationship to one another, and do not require the intervention of any higher centre to bring them into harmonious play. This fact leads him to consider the influence of the bases of the optic lobes on locomotion, and he concludes to the sensory nature of their function. Experiments are adduced to show that after extirpation of the optic lobe on one side, cutaneous excitations on the opposite side of the body are not followed by a jump, as they are on the other. It would thus appear that in the frog all the motor cerebral mechanisms are in the bulb, and that the other encephalic organs serve only for the elaboration and coördination of the afferent impulses on which spontaneity and psychical life depend.

Dr. Steiner distinguishes between the simple "excitomotor" reflexes of the cord, with the more complicated processes of the bulbar general motor centres and which he terms "sensorimotor" without wishing to imply that they are above the plane of consciousness. A frog deprived of all its encephalon beyond the medulla oblongata reacts by a jump to the application of a weak acid to the skin. When the solution is stronger, the locomotor reflex is succeeded by the more elementary movement of wiping off the offending substance. A stronger still liquid excites both actions at the same time, and the spinal reflex now interferes with, or inhibits, the higher bulbar mechanism. We may mention here that Eckhard and Steiner's experiments do not support Goltz's statement, that heating the water in which a decapitated frog is placed does not call forth any movements, as it does in the case of an animal deprived of its encephalic ganglia only.

The behaviour of normal and prepared frogs when placed in water offers several points of interest, and is discussed in a separate chapter by Dr. Steiner. He shows that it is neither skin excitations due to the contact of the liquid, nor impressions from the deeper structures due to the loss of weight, which alone start the swimming reflex; it may be both, but the water must also be in motion. The quantity of air contained in the lungs is an important factor in aquatic locomotion, and experiments tend to show that a special respiratory mechanism exists in the base of the optic lobes.

Observations as numerous as conflicting are recorded in the literature of the subject concerning the forced movements following asymmetrical lesions on the frog's brain. Dr. Steiner has gone over the whole ground systematically, and his results are particularly worthy of attention. These forced movements are of three kinds: circular, revolving, watch-hand-like. The first ("manège" movements, gyrations) consist in progression (walking, jumping, or swimming) in a circle; the second in rotation, in a corkscrew-like manner, upon the axis of the body; the third in rotatory lateral movements of the body about its caudal end as a pivot.

Every one is agreed upon the negative results of the ablation of

one hemisphere; but when we come to the *thalamus* we find some of the older authorities (Flourens, Renzi) speak of circular movements towards the side of the lesion, whereas Steiner gives as the result of very numerous and very accurate removals of that organ, the supervention of watch-hand rotation towards the opposite side. The probable solution is that, in the former case, the lesions (which lacked exact post-mortem confirmation) extended somewhat beyond the thalamus into the base of the lobe. After a time this rotation ceases, the frog remains quiet, and on excitation, performs circular excursions, also towards the sound side. Within twenty-four hours, locomotion becomes rectilinear again, and the animal is apparently in a normal condition.

Ablation of the roof of one *optic lobe* gives no result; but that of its base gives rise to well-marked gyrations towards the normal side, whether the animal be on land or in water. After a time exhaustion sets in, and the head only remains for a time depressed and turned in the same direction. These phenomena are permanent, and may be brought out for an indefinite time, all the movements being perfectly coördinated. It must be noted that if the operation is not perfectly carried out, and a small portion of the base is left *in situ*, there may be feeble circular and revolving movements towards the injured side.

Removal of half the *cerebellum*, and hemisection of the *medulla* far back near the calamus scriptorius, do not produce any motor disturbance. If, however, the anterior region of the latter organ is submitted to the same operation, muscular spasms are produced, and the frog, on being laid on the ground, jumps repeatedly perpendicularly upwards, and falls on the wounded side. Next comes a period of forced position of the head, which is rotated downwards towards the same side. The opposite fore-limb is rigidly extended to the ground. Placed in water, the frog first performs gyrations away from the lesion, then rolling movements towards it, or the rolling begins at once. All these phenomena are repeated whenever the animal is interfered with, for weeks after the operation. Further researches led Dr. Steiner to discover the exact lesion required to obtain continual rolling, as opposed to the periodical variety just described, in which there is an alternation of circular movements. It consists in a section across the thick edge of the bulb, enclosing the 4th ventricle, at the level of the trigeminus root, a little below the inferior edge of the cerebellum. It may be noticed, by the way, that no paralysis follows bulbar hemisection in the frog.

These results demonstrate that forced movements occur only after lesions of the thalamus, or lobe, or one side of the bulb; and that whilst circular and revolving motions are direct effects of the destructive lesion, watch-hand rotation belongs to the category of the indirect effects due to the traumatic irritation of one lobe. Destruction of the visual centre (roof of the lobe) produces no motor disturbance, though in the frog the optic decussation appears to be complete. The perfect coördination in swimming shows

that forced movements are unconnected with paralyses or contractures; and the fact that these movements do not arise without some external excitation, proves that the term "forced" is not to be taken in Magendie and Vulpian's sense, who assumed the existence of a kind of internal impulse within the nerve-centres. It would be more consonant with the facts to interpret these phenomena as muscular adaptations, the response to centripetal misinformation, so to speak, through disturbances in the receptive and elaborative centres.

Dr. Steiner describes the results obtained by hemisections at different levels, and in different directions of one thalamus, or optic lobe, for the details of which we must refer the reader to the original. One point is of interest—viz. that longitudinal division of one lobe is immediately followed by "manège" towards the opposite side; but within twenty-four hours the frog appears to be normal again in every respect, whereas a diagonal section produces permanent effects. These facts agree with the view taken by the author of the functions of the basal part of the optic lobes as end-stations of centripetal tracts. It will have been noticed that rolling movements always take place to the wounded side, even when combined with gyration in the opposite direction. Dr. Steiner concludes this portion of his work by showing that no forced movements follow bulbar lesions after removal of the commissural part, which he once more declares to be the only encephalic motor centre.

The experiments of Professor Fano on the brain of the tortoise (*Emys europæa*), were made contemporaneously with those of Dr. Steiner; and the results of these two observers gain in value and interest from having been made independently and under different conditions. The most striking fact connected with the cerebral mechanism of the tortoise is that when the animal is deprived of the whole encephalon, except the medulla oblongata, it performs for a considerable period, either at intervals or continuously, coördinated locomotor movements, without intervention of any external stimulus. These movements persist, though the animal may be arrested by an obstacle, and are of two types: either the opposite front and hind-legs strike out together, as in normal locomotion, or the rhythm is altered, and the hind-legs simply push the body forward, making it pivot on the shoulder-joints of the fore-limbs which have been previously placed, fully extended, upon the ground. These two gaits occasionally alternate in the same subject; but we are not informed of the anatomical or other causes to which this variation may be due. The movements of the brainless tortoise are throughout characterised by an expenditure of energy disproportionate with the effects produced; whilst moving, it holds its head and neck extended, and performs lateral movements of the tail.

The direction followed by such animals was studied by making them mark their paths on the floor. It is thus shown that they never walk in a straight line, but describe either undulatory

curves, or irregular circles to one side, as if there was always one half of the nerve-centres acting more than the other. After being shaken, the prepared tortoise always describes an "initial loop," or more or less complete circle, before resuming its ordinary mode of progression. Professor Fano also shows that in those animals which display the phenomenon of intermittent deambulation, there is no definite relation between the periods of rest and the activity during those of walking; and compares in this respect the action of the bulbar locomotor centres to that of the respiratory and cardiac centres in certain abnormal conditions.

The ordinary reflexes are more easily obtained in the hind-legs, whereas the coördinated locomotor movements are more marked and persistent in the fore-limbs. In one case of accidental injury the opposite condition was found to obtain; and here the action of the posterior extremities was altered, and of a retrogressive nature. Professor Fano was not able to obtain the same results experimentally, which reminds us of what happens in the frog after removing the anterior two-thirds of the basis of the optic lobe.

The motor mechanism of the medulla oblongata resides in the anterior two-thirds of the organ, for when these are separated from the cord, every movement ceases. A longitudinal median incision does not interfere with this activity. Decapitation is followed in the tortoise (as quite recent experiments have shown it to be the case in the duck also) with a few short-lived locomotor movements, as if the motor spinal elements had preserved an impression, as it were, of the higher coördinated impulses.

The occurrence of spontaneous movements in the brainless emys shows that we cannot generalise the results obtained on the frog and other animals, and speak of the encephalic centres as essential to their production. It also led Professor Fano to extend his experiments to other cold-blooded vertebrates; and after some failures, he found that the same phenomenon could be observed in toads (*Phryne vulgaris*, but especially *Bufo viridis*). Shortly after removal of all the encephalic ganglia, with exception of the bulb, the toad begins to walk (not to jump) with the ordinary crossed action of the anterior and posterior limbs; the latter, as in the tortoise, manifesting a slight delay. The gait is more flaccid than in the healthy animal, and obstacles do not arrest the movements. This ambulatory activity is never continuous, but always alternates with periods of rest, and persists for many days. The graphic method reveals phenomena essentially similar to those observed in the tortoise as to the paths described by the animals.

Successive transverse sections of the bulb prove that the motor mechanism is localised in its anterior portion, but seems to extend farther back than the corresponding centre in the frog, so that respiration ceases together with locomotion. The proximity of the locomotor and respiratory centres led the author to ascertain whether there is any relation between their activity in the toad as well as in the tortoise; but simultaneous tracings show no regularity in the collateral course of ambulation and respiration.

Professor Fano believes that the activity of the locomotor bulbar

mechanisms does not depend upon any peripheral excitation, is not a reflex, but rather an expression of nutritive activity in the centres. We cannot here adduce the various arguments by which he endeavours to support his view; but he seems to underrate the influence of the centripetal sub-conscious impulses from the organism itself as a possible source of excitation. At any rate, several of his remarks are quite reconcilable with the opposite explanation of his facts, whilst they receive no support from the results obtained on other animals.

Removal of the *cerebellum* does not appear to have more effect in the tortoise than we have seen to be the case in the frog; the *optic lobes*, on the other hand, appear to play an important part in the motor manifestations of the former. Their presence, after removal of the hemispheres and thalami, is sufficient to arrest the locomotor activity of the bulb; and systematic sections show this influence to be exerted by the basal portion of these organs. On the other hand, removal of the optic lobes only produces great restlessness in the movements of the animal, which, on being interfered with, no longer withdraws into its carapace, or even slackens its pace. Professor Fano distinctly asserts that tortoises can see so as to avoid obstacles, after complete ablation of the lobes; and accepts Setschenow's theory, that these organs inhibit reflex movements by retarding the transmission of excitations through their ganglionic structure. It is difficult to reconcile these observations with the results of similar experiments on frogs and fishes.

Ablation of the *hemispheres* in the tortoise does not appear to suspend spontaneous movements; the animal walks, swims, and acts as a normal specimen, only more slowly. Electric excitation of these organs, if protracted for a minute or so, is followed by a short period of active walking, during which the animal does not respond to external excitation, "as if the influence of the optic lobes had been suspended." The same phenomenon can be elicited several times in succession. Electric excitation of the thalami in the whole animal produces symptoms of pain, the limbs being violently withdrawn within the carapace. The same excitation, after removal of the hemispheres, and protracted for a sufficient period, may give rise to feeble locomotion, which becomes more pronounced upon ablation of the optic lobes.

Unilateral lesions of the brain in tortoises does not give rise to any such varied and marked forced movements as they do in the frog. Removal of one hemisphere, as usual, is ineffective; but when the corresponding lobe is taken away also, circular or "manège" movements are observed, the animal's walk being in large circles towards the opposite side. There is no hemiplegia, and the posterior extremities move harmoniously; but an alteration is perceptible in the coördination of the two anterior limbs. The lesion being on the right side, the left leg performs movements of adduction, the right of abduction, after having been placed on the ground, and the circular movements are to the left. Professor Fano does not account for these phenomena on the theory of forced movements, but assumes that there are in each hemisphere and lobe, centres for

the adduction of the leg on the same side, for abduction of that on the other. He finds that on dividing the cervical cord longitudinally along the middle line, the animal walks in larger circles, and that there is less abduction of the right leg; and he explains the latter fact by assuming a partial decussation of the motor fibres in the cord. On continuing the incision upwards, so as to bisect the medulla in its whole length, the animal starts upon its gyrations in the opposite direction; the left fore-leg continues its movements, whilst the right hind-leg is motionless, and the right fore-leg performs large ineffectual excursions, which cease on removal of the motor bulbar centre. We do not see any difficulty in explaining these phenomena on the theory of forced movements; and, without assuming the unlikely existence of adductive and abductive motor centres in the fore-brain, in accounting for the facts by an abnormal action of the bulbar centre dependent upon a disturbance in the higher receptive and regulative centres.

The general result of the most recent researches on the cerebral functions in amphibians and reptiles is thus that the greater part, if not the whole, of the encephalic motor centres is contained in the anterior part of the medulla oblongata. The hemispheres apparently contain the elaborative mechanisms forming the physical substratum of the higher conscious processes. The thalami seem to be connected with cutaneous, the basis of the optic lobes with motor, feelings. The influence of the cerebellum on coördination is but slight in the lower classes of vertebrata; the researches of Baudelot[1] on fishes had already demonstrated this fact, which the small development of that organ might have led one to anticipate. The importance of these results towards the elucidation of many problems of cerebral localisation in man can hardly be overrated; and we may look to the comparative method of investigation for much light and assistance in the future.

A. DE WATTEVILLE.

MEYNERT (THEODOR). *Psychiatrie. Klinik der Erkrankungen des Vorderhirns begründet auf dessen Bau, Leistungen, und Ernährung.* Erste Hälfte mit 64 Holzschnitten und 1 Tafel. Wien: Wilhelm Braumüller, 1884.

MEYNERT (THEODOR). *Psychiatry, a Clinical Treatise on Diseases of the Fore-Brain, based upon a study of its Structure, Functions, and Nutrition.* Translated by B. SACHS, M.D. Part I. New York and London: G. P. Putnam's Sons, 1885.

A WORK from the pen of Prof. Meynert on the Diseases of the Cerebrum is an event of no small importance, and the English reader is under a deep debt of gratitude to Dr. Sachs for placing within his reach a translation of it, which is as accurate, clear, and

[1] See 'BRAIN,' April 1884, p. 133.

readable as any translation of Meynert's writings is ever likely to be. Those who are acquainted with the obscure and involved style of the original will readily understand that Dr. Sachs' task has not been by any means an easy one, and will learn without much surprise that "the attempt at a literal translation had to be abandoned." The first half of the first part of the work—the only part which has yet appeared—deals with the structure, architecture, and minute anatomy of the brain. For nearly a quarter of a century Prof. Meynert has been instructing us in the anatomy of the brain, and much of our present knowledge of the structure of the organ is due to his labours. But much has happened during this time, and it is possible that the teaching which was new and in advance of the age in the beginning, is old-fashioned, and behind the age at the end of the period. The method of investigation adopted by Meynert—the old cleavage method—is by no means a perfect one, and it is only in the hands of a thoroughly practised expert like himself that anything like trustworthy results could be obtained by it. And although we believe the statements of Meynert with regard to the structure of the brain to be on the whole accurate, yet we have no hesitation in rejecting any of his conclusions which are found to be in conflict, and not a few of them are in conflict, with knowledge obtained by means of better methods than the one he adopts, such as the method of embryology, as practised by Flechsig; that of arrest of development, as introduced by Gudden; and that of secondary degenerations, as employed by Türck, and Charcot and his pupils.

But even after making many deductions for the shortcomings of this work, the reputation of Meynert as a great anatomist is, on the whole, thoroughly sustained in it. There are not many anatomists who would find much scope for the exercise of their talents in giving a description of the basal surface of the brain; but nowhere probably is the particular genius of Meynert seen to greater advantage than in furnishing such a description. The small print on page 26 (page 28, transl.) we regard as a peculiarly favourable specimen of his work. The author's unrivalled knowledge of the comparative anatomy of the brain has enabled him to compress in a few lines a truly astonishing amount of information. Every sentence is a generalisation, and every generalisation helps to explain the structural and functional differences existing between the human brain and the brains of the lower animals. Many pregnant passages of this kind are to be found in the work before us, and in some few places new lines of investigation appear to be opened up, which will be sure to bring a great reputation to any one who may have the sagacity to see their significance and the industry to pursue them.

The next section of the work is entitled "Anatomical Corollaries and Physiology of Cerebral Architecture;" but it really treats of the relations existing between the structure of the brain and mental processes. In order to discuss this subject profitably an author ought to be not simply a great anatomist, but a great

subjective psychologist also, his knowledge of psychology being attained either by the analysis of his own thoughts and feelings, or by the analytical labour of others. Judging from the work before us, we have no hesitation in saying that Prof. Meynert's knowledge of subjective psychology is an exceedingly slender one, and we pass upon him a very temperate judgment in saying that his psychology is fully a generation behind the psychology of this country as represented by the writings of such men as Bain, Spencer, and Hughlings-Jackson. But temperate as we believe this judgment to be, it will be expected that some considerations in its justification will be advanced. Let us take as a test the principle, or rather postulate, with which the author starts what he calls his anatomical corollaries. He asserts that "the only specific energy of brain cells is a faculty of sensation (Empfindungsfähigkeit, translated by Dr. Sachs 'sensitiveness'), which is converted in the forebrain into actual sensation." But to postulate the existence of sensitiveness or of a faculty of sensation, in order to account for the fact of sensation, appears to us to be an attempt to explain the *obscurum per obscurius*, and when Meynert proceeds to apply and illustrate this principle, the result appears to reach the superlative degree of the obscure. Surely no one is assisted in comprehending the connection existing between the activity of brain cells and sensation by being told that "in assimilating totally unknown physical impulses, the cerebral cortex and complicated protoplasmic structure, resembles the protoplasm of the primitive amœba, which can transform itself into a hollow mass, and can thus encircle any body which it wishes to assimilate." This comparison may be regarded as a good metaphor, but has no claim to be regarded as a scientific generalisation.

The next principle which Meynert advances is "that the processes of innervation from the hemispheres, which constitute what we term *volitional* acts, are nothing more than the perception, or memory of sensations of innervation (Innervationsgefühle.)" This doctrine is not very intelligible as it stands, but the context shows that by "processes of innervation from the hemispheres," Meynert means the motor actions which are set up by excitation of certain areas of the cortex of the brain, and by "sensations of innervation" he means the sensations which accompany muscular effort, or, in other words, the muscular sense. This principle, therefore, in reality asserts that the so-called motor centres of the cortex are not motor centres at all, but are sensory centres of the muscular sense. This is the theory which has been adopted by Munk, and which has also played a considerable part in the explanations of Goltz and other German physiologists. A good deal of ingenuity is manifested by Meynert in illustrating this theory, but it must still be regarded as a plausible hypothesis which has not yet received its verification in facts; and even if all the author's statements were acknowledged to be true, the only conclusion warranted by them is that the cortical sensory centres for the muscular sense coincide with the centres from which the centrifugal conducting paths for volitional movements take their

origin. Some few pathological facts can be adduced in favour of this opinion, whilst others might be adduced which are not in accord with it. None of these, however, are mentioned in this part of the work, but we may expect to find them discussed in the clinical part. But the known facts that centrifugal conducting paths emerge from certain areas of the cortex, as is indeed shown on Meynert's own diagrams; that the fibres of these conducting paths spring from large caudate cells more or less similar to the motor cells of the spinal cord; that the areas in which these cells exist give a motor reaction to electrical excitation, while the areas in which they are absent give no such response; and that destruction of the areas with the large pyramidal cells gives rise to a descending degeneration of the conducting paths, which can be traced into the spinal cord, while destruction of other areas is not followed by any such result, combine to prove in the most conclusive manner the propriety of designating areas which are endowed with such properties by the name of motor centres; and we believe that every argument which has been adduced by Meynert in opposition to this conclusion is merely so much misplaced ingenuity. But if any one is enamoured of Meynert's theory of the motor centres, he had better see how he likes it when applied to the explanation of the phenomena of disease. The author mentions the case of a boy who was affected with a large tuberculous tumour of the mesencephalon and thalamus. In addition to paralysis of the right third nerve, and of the left facial nerve and left extremities, it was noticed "that the head of this boy was turned towards the left side, and that, while at rest, there was flexion of the left upper extremity, and extension of the right upper extremity." The following is the author's explanation of this forced attitude:—"We may suppose that the boy," says Meynert, "lacking the innervation-sensations of the left rotatory muscles of the spinal column, was induced to excite the missing sensation of innervation by a volitional rotation, to produce the lacking sensation of innervation of the left arm flexors by forcing a flexion of this arm, and under similar circumstances to compensate for the disturbed sensation of innervation of the right extensor muscles by exciting a forcible extension of the right arm. The lack of innervation-sensations created delusions with regard to the position of his extremities, which in turn gave rise to volitional movements, in consequence of which he assumed the typical pathological position, the so-called forced position." The above passage is quoted from Dr. Sachs' work (p. 165), which appears to us, on comparison with the original, to be a very faithful and literal translation of the author's words; but we have to confess our complete failure to discover from a study, either of the original or the translation, any meaning whatever in this pretended explanation. To us it seems the most pronounced example we have met with for a long time of high-sounding jargon, but others may be fortunate enough to find in the passage the wisdom which is hidden from our view.

After passing such a severe judgment—a judgment which we

shall be glad to see reversed by a more fortunate or more discriminating critic than ourselves—it is a sincere pleasure to acknowledge that the author's remarks on the functions of the association system of fibres are both judicious and well worthy of study. His theory of the functions of these fibres is practically the same as that enunciated by Mr. Herbert Spencer many years ago. That theory is that the activities of the cells of the brain are the correlatives of the feelings, and that the activities of the fibres are the correlatives of the relations between the feelings. But although the theory has been stated in general terms by Mr. Herbert Spencer, his practical acquaintance with the anatomy of the brain was too scanty to enable him to connect the relation between the feelings with a definite system of fibres. This was done by Meynert as far back as 1865, when he showed that the association fibres are connected at both ends with cortical cells.

Meynert agrees with Munk in believing that every portion of the cortex of the brain subserves intellectual functions, and he condemns the opinion of Hitzig, who placed the intellectual functions in the frontal lobes. It never seems to occur to him that it is possible for both of these opinions to be true, according to the extension given to the term "intellectual functions." As we sit writing, our eyes are raised, and we judge that a book lying on the table is three feet distant—Newton judged that the earth was ninety-one millions of miles distant from the sun. Both judgments are reached by the exercise of intellectual functions, but are the judgments themselves on the same level of intellectuality? Surely not. Our judgment was the result of direct perception, and was made in all but an unconscious manner; while Newton's was the final result of an involved and intricate train of indirect reasoning, every step of which required that highest degree of consciousness, called close attention. Now if the direct judgments, which are involved in every act of perception, are to be regarded as being reached by the exercise of intellectual functions, and this is certainly the most scientific conception of the intellect, then it is highly probable that the whole of the cortex ministers to intellectual functions; but if those judgments which are reached by indirect reasoning are alone entitled to be regarded as resulting from the exercise of intellectual functions, and this undoubtedly corresponds to the popular conception of intellectual processes, then the intellectual functions, and most probably the higher emotions are, in our opinion, localised chiefly in the anterior lobes of the brain. In one word, the difference between Meynert and Munk, on the one hand, and Hitzig on the other, is one of terminology, and not of fact. Meynert is equally unfortunate in his references to the localisation of memory. "No author of the present day," he says, "would be likely to insist on one special seat of memory, for memory is the common property of all cortical cells and fibres which are able to receive and conduct external stimuli of all sorts." This assertion is, in our opinion, true; but it is such a one-sided presentation of the facts, that it can hardly be distinguished from an absolute error. We do not believe, any more

than Meynert, that memory is localised in any circumscribed area of the brain; but we do believe that particular forms or manifestations of memory are so localised. Let us take the famous American crow-bar case, in which the frontal lobes were almost completely destroyed, as an example. The man's memory for events and dates was not sensibly impaired after his recovery, but his moral character having undergone marked deterioration, it is very likely that such words as "baseness," "self-denial," "justice," and "righteousness," would not after the injury call up in his mind the same intricate associations as they did before it. It may therefore be said that the patient suffered from an impairment of memory, although to ordinary observers loss of memory was not a prominent symptom. Let us contrast the symptoms in the case just related with those of a case in which there is a spot of softening in the left hemisphere of the brain in the convolutions which border the horizontal limb of the sylvian fissure—the first temporo-sphenoidal and the supra-marginal convolutions. Under such circumstances patients suffer from word-deafness, or, in other words, spoken language ceases to have any meaning for them, and most, if not all, of these patients are the subjects of what Kussmaul calls the aphasia of recollection, a condition in which the patient is unable to name his own bodily organs, or any single object presented to him. In the face of facts like these, how futile it is to assert, without any qualification, that memory is the common property of all cortical cells and fibres!

In his Preface the author tells us that he has deemed it necessary to criticise "Darwin's theory of the inheritance of acquired faculties." The promised criticism of this doctrine is in the following terms:—"The Darwinian doctrine that ideas are inherited, and are not the result of perception and association; that movements, even mimical ones, are the result of innate motives, and have nothing to do with imitation and early reflexes, can hardly be applied to man. Not even his upright gait, which is surely a universal form of movement, is innate in man; it is acquired with difficulty only through imitation and cortical co-ordination."[1] So far as can be gathered from the present work, Meynert is an adherent of the empirical school of philosophy, and he would have us start our explanations of mental phenomena with the *tabula rasa* of Locke. But if he denies the existence of notions native to the mind, or innate ideas, then he has to combat not Darwin's views, but the whole of Kant's transcendental æsthetic, and the transcendental logic, or, at any rate, the first part of it which constitutes the transcendental analytic. He will also have opposed to him every philosopher of note since the time of Kant, with the exception of Brown, Bentham, and the Mills in this country, and possibly Comte in France, but with, so far as I know, no exception whatever in Germany. It is not, therefore, to be expected that any author, however gifted he may be, can dispose of the theory of *à priori* notions, or innate ideas, in a couple

[1] Page 256, trans. p. 274.

of sentences. But if a critical analysis of our thoughts and feelings shows that some, not all, of our ideas are *à priori* or innate, the question then arises—have these ideas been implanted in our minds by a special creative act, or have they been formed by a progressive development? The existence of innate ideas was used very effectively a few years ago in this country by the opponents of the Darwinian theory, and in all their arguments they assumed that these ideas were a specially created endowment of the human mind. And it was not Mr. Darwin, but Mr. Herbert Spencer, who advanced a theory of innate ideas, which brought them into conformity with the general doctrine of evolution. Mr. Spencer did not deny the existence of innate ideas, but he showed that, although these ideas could not have been generated by the experiences of any single individual, yet they were produced in us by the accumulated and inherited experiences of the race This doctrine has reconciled the differences between the empirical school of philosophy, which took its origin with Locke, and the intuitive school which dates from Kant, and it has produced a profound revolution in psychology, as well as in philosophy—the most profound and far reaching, indeed, which this century has witnessed in any department of knowledge; and if Meynert chooses to attack this doctrine at any future time, we hope he will grapple with it as it is presented by its original author. No one denies that individual experience is necessary in order to develop even innate ideas, just as no one denies that light is necessary to develop sight, although our ability to see depends chiefly upon the fact that we are born with eyes. And similarly, although a child requires a considerable amount of training before he can maintain the upright posture, the fact that he can maintain it with ease, after such training, results less from the training than from the structure of his body; and this, in its turn, depends upon the innate tendency the organism has to grow into the parental shape.

Nothing could show more convincingly the utter incapacity of Meynert to deal satisfactorily with subtle psychological problems and philosophical discussions, than the manner in which he jumbles in inextricable confusion such words as "the ego," "the individuality," and "the character." The ego, according to Descartes, is the thinking substance in man, in contrast with the extended substance of which his body and the surrounding world are composed, and the word has been used in this sense by almost all subsequent philosophers. Most philosophers would refuse the possession of an ego to the lower animals, and we know of no author that would accede the possession of it to vegetables. Individuality, however, belongs to vegetables as much as to man, and indeed, some biologists believe that the individuality or the principle of individuation is coextensive with living beings, and they have accordingly included the principle in their definition of life. In its primary meaning the word "character" refers to the moral qualities of a man, and in its secondary meanings, a good or bad character is given to animals, vegetables, and even to inanimate objects according as they are useful or injurious to man. It will

thus be seen that "the ego" is a philosophical term to indicate the mind in opposition to matter, that "the individuality" is a biological term which refers to all living beings; and that "the character" in its primary meaning, is a term of moral science, although in its secondary meanings it has a more extended application.

Let us now listen to what Meynert has to say with regard to these terms. He believes that there is a primary ego which is defined by the limits of the body, but he continues, "the primary ego expands through permanent and intense secondary perceptions joined to it by association; so that intimately related persons, property, skill obtained by constant practice in art, science, a fondly cherished aim in life, convictions, patriotism, and honour become part of the ego" (p. 175, trans.). When speaking of the cortical centres, he says—"the sum of these 'centres' constitutes 'the individuality,' the 'ego' of abstract-psychologists;" and a little further on he adds, "this unequal activity of the fore-brain, constituting individuality, varies as regards contents and degree with each person; it is designated also the *character* of the individual" (p. 167 et seq., trans.). Our wealth of philosophical language is not so great that we can, without offering the strongest protest in our power, allow words which have had definite meanings assigned to them for generations to be sacrificed in this rude manner by having their differences effaced, and their connotations reduced to an identity.

In the next section of the work Meynert treats of the "Nutrition of the Brain," and his remarks on this subject will be found to be less obscure and much more worthy of consideration than anything we have met with in his psychological discussion. In his theory of disordered nutrition, however, he attaches much too great importance to the action of the vascular system with its nervous mechanisms, and too little to the action of the nervous tissues which are the seat of the abnormal changes.

The first part of the work is brought to a close by an Appendix on the "Mechanism of Expression," and of it we shall only say that the readers of Darwin, Spencer, and Bain will gain nothing, but will lose much by adopting Meynert's interpretations. An author who rejects altogether the theory of "the inheritance of acquired faculties" cannot be expected to throw much light on the mechanism of expression, and accordingly Meynert does not afford us much enlightenment; but on the contrary, he casts the haze of his heavy and obscure style over this subject, which is all the less excusable inasmuch as it is treated with such pellucid clearness in the charming pages of Darwin.

JAMES ROSS.

Illustrations of Unconscious Memory in Disease, including a Theory of Alteratives.—By CHARLES CREIGHTON, M.D. 1 vol. 8vo. pp. 212. London: Lewis, 1886.

THIS work of Dr. Creighton constitutes an important addition to our stock of pathological knowledge, it assists us in the interpretation of many morbid processes, and will no doubt be further developed in the hands of the author. The term "unconscious memory" is one not altogether unfamiliar to us; modern research into such subjects as instinct in animals, the automaton action of infants as well as several acquired habits of adults, have led us to look upon many complex movements as the result of mechanisms without any aid of thought or consciousness. If such an act as playing the piano may occur whilst the mind is wandering, and the performer scarcely conscious of it, we cannot avoid the conclusion that the memory is still awake, and therefore the appropriateness of such an expression as unconscious memory. The somnambulist may perform some wonted act and not be conscious of it; in this case the memory cannot be said to be lost. In the same way a morbidly acquired habit may persist long after all consciousness of the first voluntary act has passed. In the case of a young animal choosing its food, it may be said that the memory of a like act was transmitted by its predecessors, and there are some strong upholders of Darwinian doctrine who would account in this way for all inherited instincts. Instead of saying that instinct guided it, a more intelligible expression would be that the animal retained a memory of the past.

For some years we have employed the expression in the instruction of students, having been more especially indebted for the use of it to the author of "Life and Habit," a gentleman, it will be remembered, who contended for the priority of some of the ideas enunciated by Darwin. He would not say, for example, that instinct compelled the chick to break its egg at a certain period of its development, but that the occupier of the shell remembered the particular day and manner in which its parents broke through their covering, and made their escape. We have thus found the term very useful in the explanation of the continuance of many diseases, especially those of the nervous system. An acquired habit in an individual is nothing more than the education of a spinal centre for a particular act or to a new function, and in this sense habit is second nature. A choreal movement which lasts during life may be regarded as a newly acquired function of some part of the nervous system. An analogous condition of the sensory centre would afford an explanation of a pain which was altogether subjective. If motor cells, instead of being quiescent, had been taught to act rhythmically and regularly like those of the respiratory centre, a new and peculiar movement would result; and, in a similar manner, if some central sensory cells remained in a polar condition after the natural distal stimulus which excited the

action was removed, the pain would nevertheless continue, and be styled in ordinary language subjective.

Having already had our mind directed towards some of the propositions and theories propounded by Dr. Creighton, we were the better prepared to consider them than if they had dropped upon barren ground as a pure subject of speculation, and thus we can with the more confidence give our support to this most valuable contribution towards an explanation of many of the abnormalities of the human body. The doctrine is a distinct offshoot of the evolutionary and Darwinian theory, showing how fruitful has been the new departure of thought in every department of science.

The author has adopted the term from Hartmann's "Philosophy of the Unconscious," and from Hering's work on "Memory as a General Function of Organized Matter." Dr. Creighton's object is to show how a large number of pathological states are explicable on the idea of the continuation of the memory of past conditions or actions, whether these be in nerve centres or in secreting organs. He gives a large number of examples where a morbid state is perpetuated when once started into action, and even when the original cause is removed, and out of this highly scientific and philosophic idea, speculative as some may think it, a highly practical result ensues, and in this respect Dr. Creighton's book may rival many works on pure therapeutics; the theory of cure being simply this, that a tissue has taken on a morbid or unusual habit, and the medicine breaks the habit. Herein lies the explanation why so many different remedies may be found for curing or averting disease. We might take, for example, the cure of phthisis by various remedies, such as altered diet, a sea voyage, or residence on an alpine height. None of these means could act otherwise than breaking up the tendency to morbid action in the lungs by altering all the circumstances which first promoted it. If a young man shut up in an office and surrounded by various unfavourable conditions falls into ill-health, a change of his environments would arrest the morbid process which had begun. This may be true of many other complaints as well as of phthisis; the remedy, change, being especially enforced in this case, because it had been thought that the disease being one of the lungs, the most essential element of cure was to be found in good air. Various diseases are thus cured by breaking up the habit which was fast tending to develop them. The author's theory recalls to mind D'Alembert's notion of the operation of medicine; that nature is fighting with a club, when the doctor steps in and fights with his club; the kill or cure depends upon who has the mastery. Dr. Creighton, in like manner, thinks that the so-called alterative medicines set up such a commotion in a part of the body which is not acting normally, that after the disturbance is over, it may then go on right again and pursue its legitimate course. In some cases the medicine may produce a corresponding action with the existent morbid one, and in this way drive it out according to the homœopathic doctrine; in other cases the medicine may set up

an opposite action, and get rid of the old process by mere antagonism; in still other cases the kind of disturbance produced by the drug is not clear, but the result is that good eventually comes out of it. It is sufficient to break the habit which has made some part of the animal machinery work in an abnormal manner.

The therapeutist, therefore, as well as the pathologist, will hail Dr. Creighton's work as affording him fresh encouragement for the use of drugs. For, as the author mentions, the cause of a malady may be known, and be actually removed, and yet the old habit remain until some powerful drug come into play to rouse the morbidly-acting tissue, and make it shake off the newly-acquired process. The strongest argument in favour of drugs may be found in the fact, that nothing but some very powerful agent can arrest many diseases, or, as they may be called, morbid habits.

Much more is to be found in Dr. Creighton's work than our time or space will allow us to allude to, therefore it should be read not only by those who are anxious to add some fresh knowledge to a special subject, but all who are interested in the scientific advance of medicine as it advocates fundamental truths, which lie at the very foundation of our science and art.

As before remarked, it is an offshoot from the modern mode of thought which has tended to promote scientific advance in every direction. Of this Englishmen may be well proud, for although in a large number of departments of science England is overwhelmed by the crowd of continental workers, yet our country has always stood foremost in the production of original thinkers and of men who have given a new point of departure in modern discovery. The last twenty or thirty years has witnessed a complete revolution in the mode not only of considering inorganic nature, but of the organic world also, including man and all his attributes. For this, civilization is chiefly indebted to such men as Darwin, Herbert Spencer, and Lyell.

SAMUEL WILKS.

Klinische Psychiatrie; specielle Pathologie und Therapie der Geisteskrankheiten. By Dr. HEINRICH SCHÜLE, of Illenau. 3rd ed., 534 pp. Leipzig: F. & W. Vogel, 1886.

THE two former editions of this book appeared as a volume of Ziemssen's 'Encyclopædia.' This is an almost entirely new book, the author's object being to give an exhaustive description of the clinical symptoms of insanity, including their causes, prognosis, and treatment; whereas the subject of pathological anatomy is only dealt with in the chapters on general paralysis and idiotism.

Dr. Schüle's classification of mental diseases is founded upon a new principle; he maintains that we have to distinguish them according to the condition of the brain at the time of the onset, inasmuch as the resistance of the brain depends upon whether the organ is in a "healthy" (*rustig*) or an "invalid" condition. From this point of view the author establishes the following system:

I. Mental Disease of the Developed Brain.

1. *Diseases of the "Healthy" Brain (Psychoneuroses):*
   (*a.*) Melancholia.
   (*b.*) Mania (mitis and typica), with secondary insanity (dementia).
2. *Diseases of the "Invalid" Brain (Psychoneuroses):*
   (*a.*) Mania gravis, Furor.
   (*b.*) Paranoia (acute, chronic, atonic—Kahlbaum's Katatonia.)
   (*c.*) Acute primary dementia.—Appendix: Hallucinatory stupor.
   (*d.*) Hysteric, epileptic, hypochondriac insanity.

Appendix: (*a*) Periodic, circular, alternating insanity; (*β*) insanity caused by extra-cerebral disease (puerperal insanity, &c.), and intoxication.

3. *Pernicious Exhaustion of the Brain:*
   (*a.*) Acute exhaustion—delirium acutum.
   (*b.*) Chronic exhaustion—dementia paralytica.
4. Insanity produced by organic brain-disease: modifications of progressive paralysis.

II. Mental Disease on the Basis of Morbid Disposition, or Defective Constitution.

   (*a.*) Hereditary neuroses—Appendix: Transitory insanity.
   (*b.*) Simple form of hereditary insanity—Maladie du doute et du toucher, &c.
   (*c.*) Primary paranoia—Delusions.
   (*d.*) Moral insanity (degenerative hereditary insanity).
   (*e.*) Idiocy.

It hardly needs to be said that this system, like all other systems, has its deficiencies; so we find "mania" among the diseases of the healthy brain as well as among those of the invalid brain; the type of primary (originary) paranoia is severed from the other forms of the same clinical character; nevertheless Schüle's classification opens many new points of view which are of great value for the estimation of the different forms of insanity, especially for their prognosis. The greatest difficulty will always be to find out in a given case whether one has to deal with a "healthy" or an "invalid" brain; the determination of this point must often be a matter of mere individual impression.

Dr. Schüle's descriptions of clinical cases are most instructive, full of life, the result of long and careful observation; the author's desire to avoid the conventional phraseology of text-books gives his style a peculiar stamp, but it must be recognised that this new edition of the book is very much more readable than it was in its former shape.

It is impossible to give here a detailed account of the contents of the book; may it suffice to say that in our opinion Schüle's 'Klinische Psychiatrie' is the most valuable work on the subject which has been published in Germany since Griesinger's classical

work: 'Die Pathologie und Therapie der psychischen Krankheiten;' and that we strongly recommend it to the attention of our readers. R. H. PIERSON (Pirna, Saxony).

Allgemeine Diagnostik der Nervenkrankheiten.—By Dr. P. J. MOEBIUS. 1 vol. 8vo., 338 pp. Leipzig: F. & W. Vogel, 1886.

THIS book is intended to serve as an introduction to the study of neuropathology, and it will be found very useful for the purpose. The standard works on the diseases of the nervous system require the knowledge of the different methods of examining a patient with regard to the condition of his nerves, but they do not teach the student their application in a sufficiently exhaustive manner. Therefore, Dr. Moebius's treatise, which is a plain, easily intelligible and, at the same time, thorough exposition of the subject, cannot fail to meet with much approval.

After a more general instruction concerning the way of examining a patient, taking the family history, &c., the author describes the methods of testing the state of the mental faculties, the condition of speech, chiefly after Kussmaul and Lichtheim, including the disturbances of reading and writing. Then follows the examination of the motor apparatus: active mobility, reflex, mechanic and electric excitability (the latter part after Erb's Electrotherapie). The next chapter is devoted to the examination of the state of the special senses: sight, hearing, smell, taste, and a more detailed account of the different ways employed for stating the condition of sensibility. We quite agree with the author when he finally says that for this purpose the simplest method is the best, and that all those complicated tests which are indispensable in physiological research are more or less unavailing at the bedside.

In an Appendix, the physiology and pathology of muscles, and the disturbances produced by lesions of the different nerves, are treated with much conciseness and lucidity.

Numerous excellent diagrams, partly original, but to the greater part taken from the works of Erb, Duchenne, Charcot, Strümpell, and others, add considerably to the value of Dr. Moebius's book, which is not to be considered as a mere compilation, but shows throughout the hand of a critical and experienced observer.

We gladly take this opportunity to mention, that with the present year Dr. Moebius has assumed the joint editorship of *Schmidt's Jahrbücher*, the 209th volume of which is in course of publication. His name is a token, that neuropathology will henceforth be worthily represented in the familiar pages of this old-established record of medical progress.

R. H. PIERSON.

Abstracts of British and Foreign Journals.

S. Kostjurin on the Senile Changes of the Cerebral Cortex (*Wiener Medic. Jahrbücher*, 1886, Hft. 2).—The author examined a number of brains of old people in Professor Obersteiner's laboratory, in order to establish the histological changes to which the various tissue-elements of both simple and senile atrophy may be referred.

He arrived at the following conclusions:—

1. The great part of the nerve cells in old age belong to a class of a more or less well-marked pigmented fatty degeneration, and show sometimes the formation of vacuoles.

2. The medullated nerve fibres of the cerebral cortex in part atrophy and become fewer in number.

3. The small blood-vessels are affected by atheromatous degeneration, viz. an almost universal spindle-shaped thickening of the connective tissue of their walls, which may result in the entire closing of their lumen. The quantity of the normal amount of pigment present in the adventitia of the arteries seems slightly increased.

4. In place of the atrophied cells and nerve fibres, there is a slight thickening of the connective tissue.

5. At the periphery of the convolutions of the brain there are always a larger or smaller quantity of corpora amylacea, which may form a continuous enveloping layer round the cerebral cortex.

These several degenerative changes are, as regards their intensity, not so dependent on the age of the individual, but rather correspond to the decrease of weight of the brain; in heavy, though at the same time very old brains, they may be nearly absent, whilst in much younger but particularly light brains, they are very marked. This is mostly the case when there are degeneration and decrease of the nerve fibres.

Further, the above described changes of the different elements forming the cerebral cortex will suffice to explain all those phenomena which characterise senility, as far as the activity of the brain is concerned.

One must regard them as the normal result of the brain of old age, and when other pathological appearances are present in such brains they must be referred to other causes than old age.

L. Merk on the Arrangement of Karyskinetic Figures in the Central Nervous System and Retina of Embryos (*Sitzungsberichte der R. Acad. d. Wissensch. in Wien*, 92 B., 1885).—In embryos of the Tropidonotus natrix one finds in the earlier stages the surface of the neural tube, which is turned towards the central canal of the spinal cord, as well as the brain ventricles, studded with karyskinetic figures, whilst in the remaining wall of the central nervous system there are at that period hardly any appearances of karyskinesis to be seen. An important circumstance is that the two new nuclei almost always lie close together, not radiating one behind the other, and one would almost conclude that the embryonal central vesicles are by this means enabled to retain their own form, the nuclei acting like the keystones of an arch. In addition to this (if karyskinesis is the only way in which cells and nuclei divide themselves in the brain), the conclusion is more certain, that the epithelium of the ventricles represents the matrix for the whole wall of the brain, at least during a considerable period of embryonal life. The same appearances are seen in the retina.

H. Kaan on the Relation between Hypnotism and Cerebral Circulation (Wiesbaden, 1885).—When a warm fomentation is applied to the head of a person in a state of hypnotism, the patient is aroused at once; but with a cold compress, the hypnotic condition is increased. On the other hand, in catalepsy a cold compress produces the lethargic condition. The author concludes from this, that in these two states the conditions of blood supply to the brain are diametrically opposite. He accepts, with Heidenheim, that in the monotonous, prolonged excitation of the senses in the process of hypnotising, there ensues a reflex abnormal anæmia of the brain (lethargy), which, if the excitation be continued, is followed by an equally abnormal hyperæmia (catalepsy). This conclusion is borne out by the variation in the volume of the arm, which was observed by means of Mosso's plethysmograph. In the lethargic stage there was an increase of the volume-curve for the arm (corresponding to the anæmic state of the brain), and in the transition to catalepsy a diminution of this curve was seen. PROF. OBERSTEINER, M.D.

Paneth on the Cortical Motor Area of the Dog (*Archiv f. Physiologie*, p. 523, vol. 37, 1885).—The author's experiments were carried out in the laboratory, and under the supervision of Prof. Exner. He used an apparatus constructed so as to furnish galvanic currents of fixed duration and graduated strength in the exploration of the cortex of narcotised dogs. He first determined the excitable areas with reference to certain muscles, especially those of the feet, one of which—the extensor digitorum, for instance—being selected, and attached to the lever of a myographic cylinder. The corresponding excitable area was next circumscribed by means of an incision, which left it connected with the subjacent parts only. Now if there are any fibres establishing direct communication between the latter and the cortex, the effects of electric excitations must remain the same as before; and this too was found to be the case of course. When such fibres (the direct pyramidal tract) were also divided, no effect was obtained by a moderate current. So far Dr. Paneth confirms in every particular the position held by most physiologists at the present day. The original portion of Dr. Paneth's work is his attempt, by means of circumscribing cuts, to isolate a portion of cortex, stimulation of which should cause movements of one limb or one group of muscles only. This, he expressly states, he failed to do, and as his experiments seem to have been performed carefully, this is an important result. Too much stress must not be laid upon it, however, since a circumscribing cut through the cortex is almost immediately filled with blood, and so spread of the current to neighbouring parts is not prevented. The exact determination of representations of groups of muscles surely is not possible in such an imperfectly evolved cortex as that of the dog, and under any circumstances the problem would be more easily solved by attacking lower and less complicated centres. Dr. Paneth does not appear to be aware of Dr. Mariques' recent work in the same direction, viz. the isolation of motor centres from the neighbouring cortex, which was reviewed in the last number of 'BRAIN.'

VICTOR HORSLEY.